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REPORT OF SIX UNUSUAL CASES OF SEPTIC SINUS THROMBOSIS IN CHILDREN. OPERATIONS. ONE DEATH.*

DR. SAMUEL J. KOPETZKY, New York.

Introductory. The following report of six cases of septic sinus thrombosis occurring among children is made because they presented clinical pictures and offered problems unusual in every-day practice. Yet the conclusion is inevitable that similar cases occur more often than is apparent from a perusal of the literature. They are all unusual in a different way.

In the first place, they show that not only the otologist, but also the medical attendants of such patients, must have a more detailed knowledge of mastoiditis, and at least a comprehension of the various types of mastoid infections. The case histories of Cases II, V and VI showed one of the most insidious types of mastoiditis—the painless type. In this series, only in Case II was it accorded the recognition its gravity warranted. In Cases V and VI, it was at first entirely unrecognized.

Fortunately, not all cases of painless type of mastoiditis develop sinus thrombosis. In considering the prevalence of this type of middle ear infection, therefore, a larger number of such cases could be presented to stress the gravity of this lesion. The writer has seen many children, and a smaller number of adults, presenting this type of lesion, in whom diagnosis was entirely missed. They had

been considered as purulent otitides until sometimes chance, or the impatience and anxiety of the parents of the patient, brought other medical counsel, and they were then brought to operation and cure. Case V, which terminated fatally, and a case in an adult previously reported,* show the whole course of the disease when unrecognized early and when operated upon too late. Case VI of this series shows a similar case, operated upon after the otitic sepsis had become so well established as to nearly cause the loss of the patient.

A second point upon which emphasis might be placed is the belief held among the laity, and to some extent among physicians, that a timely mastoidectomy prevents the formation of a sinus thrombosis. This belief needs revision in the light of a larger experience with these intracranial infections. In the so-called coalescent type of mastoiditis,** where an interval of time is necessary for the evolution of the lesion, and where the disease spreads from the bony mastoid structures by contiguity to the sinus wall, such a belief has some foundation. But in a second group of cases, where the lesion is of the so-called hemorrhagic type, this is not the rule. Here other factors are called into being. It is not the writer's purpose to enlarge upon those factors here. They have their bases in the embryonic similarity of the tissues lining the structures of the ear and those of the upper respiratory tract, and in the specificity of action of the streptococcus hemolyticus, which is the common factor in the etiology of the influenza attack which these patients suffered, and of the otitides which subsequently developed, complicated by the blood vessel lesion.

Case I. Sinus Thrombosis Involving Jugular Bulb—Pain Over Frontal Sinus from Pressure of Enlarged Glands Upon Temporo-auricular Nerve—Repeated Negative Blood Cultures—Operations—Recovery.*—On December 12, 1919, E. F., female, aged 6 years, was admitted to the Beth Israel Hospital.

Three days before admission, the child awoke from sleep with a temperature of 104°, complaining of pain in the right ear. Paracentesis was performed, following which the patient ran an up and down temperature ranging between 99° and 104° until admission to the hospital. During all this time the child com-

*Intracranial Infections. The Laryngoscope, December, 1919. Case IV.

**Sinus Thrombosis Accompanying Middle Ear Infections. American Journal of Surgery, May, 1920.

*Patient was presented, Otological Section, New York Academy of Medicine, February, 1920.

plained of earache and pain behind the right ear, radiating to the back of the head. There was a discharge from the ear; the micro-organism from the ear discharge was the streptococcus.

Two years before the child had been operated upon for acute mastoiditis on the left side; but, except for this, she had never been seriously ill.

Just prior to admission, the child was seen by Dr. Oscar M. Schloss, who concurred in the diagnosis of acute mastoiditis.

On admission, physical examination was negative except for the scar of the left mastoid operation and the signs of acute mastoiditis in the right ear. The patient complained of tenderness on pressure at the tip of the right mastoid; there was no swelling nor redness over the mastoid area. The temperature on admission was 101°, pulse 116, and respirations 26. In the afternoon the temperature rose to 101.8°, pulse to 136, respirations to 30. The child looked more acutely ill and prostrated than the local signs warranted.

At operation it was found that the mastoiditis was of the hemorrhagic type, with thin pus scattered throughout the mastoid cells. The operation was completed with no exposure of the lateral sinus or dura.

Following the operation, the child had an uneventful convalescence. For four days the temperature ranged between 99° and 100°, and on the fifth day it was normal. On December 18, the child was discharged, six days after operation.

On December 19 she had an attack of tonsillitis (diphtheria). There were discreet spots of membrane on the tonsillar stumps (the tonsils having been removed about three years previously), and, because of the suspicious character of the membrane, an injection of diphtheria antitoxin was administered. There was a negligible reaction and the local lesion promptly cleared. The patient, however, ran a low septic temperature until December 21. From this time, however, until January 8, 1920, she did well and reported at the office regularly for dressings.

On January 8 the temperature suddenly rose to 104.6°, and dropped on the morning of the next day to 99°. On the 10th it rose again and reached 105.4°. The mastoid wound was still clean; the principal complaint was headache and a pain in the region of the ear, most intense above the line of the lower jaw. The swelling, which appeared about this time, seemed to involve the higher lying cervical glands, commencing at the glands situated between the anterior border of the mastoid process and the

ascending ramus of the jaw. The skin over these was tense and painful to pressure. No fluctuation could be determined. The boundary of the swollen mass was indefinite, and not sharply marked. Lower down the neck, the chain of glands along the tract of the internal jugular vein could easily be felt, and were found to be progressively less painful downward toward the clavicle as they were palpated.

A blood culture taken on this day was subsequently reported sterile. Differential blood examination revealed the following: Hemoglobin, 90 per cent (Kuttner); erythrocytes, 4,500,000; color index, one; nucleated reds, none; anisocytosis, none; poikilocytosis, none; polychromatophilia, none. The total count was 21,400 leucocytes, of which 89 per cent were neutrophiles, 10 per cent small lymphocytes and 1 per cent large lymphocytes.

In the effort to find the cause of the temperature and the headache in the presence of a negative blood culture, a lumbar puncture examination of the cerebro-spinal fluid was made, and 15 to 20 c.c. of spinal fluid were removed, coming away under increased pressure. The appearance of the fluid was clear and watery, with a few floculli. Albumin and globulin were normal, and sugar reduction was present. The cell count showed five cells per c.mm. A smear of the sediment showed 100 per cent lymphocytes, no polymorphonuclears being present. The bacteriology was negative, and after a two-hour search no tubercle bacilli were found.

Examination of the urine showed it to be clear in appearance, of an amber color. There was a highly acid reaction, and a very faint trace of albumin. There was no sugar nor indican. The acetone and diacetic acid reactions were two plus. Microscopic examination showed that there were a great many leucocytes present, but no red blood cells. There were a few epithelial cells and a moderate amount of pus.

During the ensuing four days, the temperature gradually dropped to normal, but the clinical picture grew worse. The swelling along the jugular tract increased in size and seemed to interfere with the patient's ability to swallow. When forced to do so, the jaw could be moved and the mouth closed and opened, but the patient elected to hold the jaw fixedly open, with the tongue slightly protruded, and continuously cried and moaned with pain. The inability to take nourishment even of the liquid kind became a factor of clinical importance.

Meanwhile, local examinations of the pharyngeal wall, the tonsillar fossae (tonsils had been removed some years previously, and

new lymphoid tissue excrescences were in evidence as replacements) gave negative results. The pain from the region of the swelling, radiating over the face, gave a point of intensity over the frontal sinus on this side, and painstaking examinations and radiograms disclosed negative findings.

On January 14, 1920, the temperature again rose to 104.6° and the patient was readmitted to the Beth Israel Hospital on January 16, 1920.

Upon admission she seemed acutely ill, with face flushed and mouth gaping. There was no tenderness on the head except over the mass in front of and below the right mastoid region. The pupils were equal and reacted to light and accommodation; and there was no nystagmus nor strabismus. The jaws were held apart and the patient could not close them without evidencing severe pain; but the temporomaxillary articulation was not tender to pressure. There was a systolic murmur heard over the precordium, but the quality of the pulse was good. Otherwise, the examination was negative.

On admission, the temperature was 103.6°, pulse 136, and respirations 20. A blood count showed a total of 11,600 white cells, with a differential count of 61 per cent polys and 39 per cent monoes. There was a marked trace of albumin in the urine, but no casts. A blood culture taken at this time was later reported negative.

The next day, January 17, the temperature rose to 105.2°, pulse to 150 and respirations to 32. A blood count showed a total of 22,800 leucocytes, of which 88 per cent were polynuclears and 12 per cent mononuclears. There was now presented a case for diagnosis, and a question of procedure.

The idea of a septic sinus thrombosis had been before the medical attendants from the commencement of the illness, but the lack of substantiating evidence in the form of a positive blood culture had deterred operative interference. Various explanations for the pain and glandular infection were advanced, and seemed unsatisfactory. The patient was rapidly losing ground.

A conference with Drs. Robert J. Carlisle and Isadore Friesner determined that heart and lungs were negative; and, in view of all other negative findings, it was determined upon to explore the lateral sinus and ligate and resect the internal jugular vein.

Operation was immediately undertaken. The neck was operated upon first. Upon exposing the vessels in the neck, the carotid sheath was found covered with large lymph nodes which obscured the field. They were adherent to the jugular vein, and made the operative work extremely difficult. The vein was finally separated and re-

sected. It was found to be very friable, and when the ligature tying the upper end of the vein slipped, it was found impossible to grasp the bleeding end without repeatedly tearing it. An incision was therefore made higher in the neck, and the vein ligated. The wound was closed, a rubber tissue drain being inserted into the lower angle.

The mastoid was then curetted free of its granulations and the sinus exposed from bulb to knee. The sinus wall was found thickened, plugs were placed above and below, and the vessel was incised between them. There was only a sluggish flow of blood when the lower plug was removed, which became more free after a curette had been introduced into the bulb and the region cleansed of clot. There was an adequate flow of blood from the cerebral side. The mastoid wound was then packed in the usual manner, and retention bandages applied.

The operative findings indicated a thrombus in the jugular bulb extending upward into the lateral sinus.

The patient was gradually going into collapse, the pulse during the operation became extremely thin and rapid, and an intravenous of 300 c.c. of saline was given on the operating table, from which the patient responded well.

During the night following operation, there were repeated periods of collapse, but eventually the patient responded to stimulants and sustaining nursing.

The day following, postoperatively, the temperature dropped to 100°, but rose immediately to 104°, pulse to 150, and respirations to 28. The child was restless, kept her mouth wide open, and still complained of excruciating pain at the angle of the jaw on the right side. She would make no attempt to close her mouth, and was apparently in agony when pressure was exerted on the mandible. The throat and teeth were normal; there was still no tenderness over the temporomaxillary articulation.

A blood count taken on the following day showed a total of 32,800 white blood cells, of which 88 per cent were polys and 12 per cent monoes.

For the next four days, from January 20 to January 24, the temperature ranged between 101°, its highest point, and normal. During this period, the same pains on moving the lower jaw were present, practically unrelieved at any time. The wound in the neck showed infection, and there was a large amount of pus on the dressings from the rubber drain. On January 20 a blood count showed a total of 13,000 white blood cells, of which 77 per cent were polys and 23 per cent monoes. A blood count made the following day showed a

total of 15,800 leucocytes, with a differential count of 69 per cent polynuclears and 31 per cent mononuclears.

Meanwhile the skin seemed more tense over the swelling in the upper neck region, and, on January 24, the entire incision was opened wide. This procedure was immediately followed by complete relief of the jaw symptoms and the pain—the pain on opening and closing the mouth disappeared almost instantly, and that over the frontal sinus also disappeared. The patient was reported as comfortable. A blood count on this day showed a total count of 9,000 white blood cells, with a differential of 67 per cent polys and 33 per cent monoes.

For the next three days the temperature ranged between normal and 100°, and from January 27 to February 4 it remained normal.

On February 4 the child was discharged. The wound in the neck had practically closed, and the mastoid was granulating.

Since then this child has been repeatedly seen by the writer and is fully recovered. The thick keloid-like appearance of the scars in the neck was greatly improved by X-ray therapy given by Dr. William H. Meyer, and when last examined, September, 1920, the wounds all looked well, even from a cosmetic standpoint.

COMMENT.

This case presents several unusual clinical findings. Septic from its very onset, had the blood culture been reported as positive for streptococci, the clinical picture would have denoted a typical case of primary bulb thrombosis. Dr. Schloss, with whom I saw the patient, was also of this opinion, but because of the absence of a positive blood culture, it was determined to explore the mastoid process and await the outcome.

The postoperative period, after the simple mastoidectomy, was so uneventful that this opinion was sidetracked. Then came a second period of sepsis, with a suspicious looking throat which subsided promptly upon the administration of diphtheria antitoxin.

The septic clinical picture which subsequently developed, with headache and markedly intense pain over the frontal region, and, eventually, the swelling of the lymph glands of the neck, was most unusual. The presence of the pain is explained by pressure exerted probably upon the temporoauricular nerve, a nerve of sensory fibres (Piersol). The immediate relief when the wound in the neck was opened and the pressure relieved was striking.

Noteworthy is the finding of negative blood cultures repeatedly, and yet there was unmistakable evidence of a clot in the dome of the jugular bulb.

From the otological surgeon's standpoint, it is to be noted that the inner mastoid tablet of bone covering the sigmoid sinus was not removed at the primary mastoid operation, there being no exposure of the sinus wall at that time. At the second operation the vein was normal in macroscopic appearance.

The question of primary bulb thrombosis will be more fully discussed under Case IV, which presented a more classical symptom complex. Here it is to be stressed, however, that no matter how strongly the suspicion of primary bulb thrombosis was entertained, yet there lacked substantiation to warrant such a diagnosis absolutely. It must be comprehended that, even with negative general physical signs from the chest, not sufficient time had elapsed for a possible central pneumonia to become evident. Hence the management of the case in such a way that the primary foci within the mastoid process were first eliminated, and surgery upon the blood vessel was done at a second and later period.

The exploration of the sinus was determined upon in the face of repeated failures to obtain a positive blood culture because the patient was steadily and rapidly losing ground. In regard to this, it may be stated that, in the finding of streptococci in the blood stream, there is substantiation of a dignosis of sinus thrombosis, all other sources of the infection being eliminated; but, in the event of negative findings, then it is better to operate upon the clinical findings than to let pass the time when surgical intervention will result in a successful issue of the case.

Case II. Sinus Thrombosis Following Acute Mastoiditis in a Patient Suffering from Chronic Endocarditis—Differential Diagnosis—Operation—Recovery.*—On January 7, 1920, M. A., male, aged 8 years, was admitted to the Beth Israel Hospital.

Eight days before admission, December 30, 1919, the child developed an attack of acute mastoiditis in the right ear, which followed a severe attack of follicular tonsillitis.

The membrana tympani had been promptly incised and a very profuse otorrhea had become established. The tenderness over the mastoid area was moderate. The examination for bacteria in the ear discharge proved the infection to be streptococcic. Cultures to determine the exact nature of the organism had not been done at this time.

The classical signs of acute mastoiditis were not present except for the inordinate amount of discharge from the ear. There was no

*Patient was presented, Otological Section, New York Academy of Medicine, February, 1920.

pain, there were no chills, and, excepting the initial rise of temperature, the fever curve was negligible. The child was languid, seemed acutely prostrated and looked ill, complaining mostly of headache and inability to sleep. Toward the end of the week, just prior to admission, there was an increase in the intensity of the mastoid tenderness evoked upon pressure.

A factor which developed at the initial examination and which was considered negligible at that time, but which, in the subsequent run of events, became of vital moment, was the finding of a systolic murmur at the apex transmitted toward the axillary line. It developed upon close questioning of the father, who is a physician, that some years previously the heart murmur was found after the child had had an attack of diphtheria, which had been treated in the usual way with administration of antitoxin. The heart was compensated and had never given particular trouble.

The ear condition was classified as belonging to the so-called painless type of mastoiditis wherein no pus retention occurs because of free and adequate egress of the purulent exudate through the drumhead perforation, and mastoid operation was recommended.

The temperature on admission was 100.8°, pulse 110, respirations 26.

The operation was performed a few hours after admission.

The mastoid lesion was of the hemorrhagic type, with some frank pus in the mastoid cells. There was more pus found as the region of the lateral sinus was approached. As the lateral sinus was reached, in its upper area near the "knee," a perisinus abscess was uncovered, the pus bathing the sinus wall being thick in consistency and yellow in color. The sinus wall as uncovered was found covered with granulations for a distance of 2 cm., coincident with the area of the abscess where the bony sinus wall had eroded away. The sinus did not pulsate, and it was freely exposed from the knee downward to the posterior bony auditory wall.

The bacteriological examination of the pus from the mastoid contents showed a mixed infection of staphylococcus and streptococcus.

At this time, while the sinus wall and the absence of pulsations made the operator think seriously of examining the sinus, it was determined to await events because the patient had exhibited no septic symptoms. Nor had the blood been cultured at this time. The wound was therefore packed lightly and the patient put to bed.

The immediate postoperative period was uneventful, except for the fact that the patient failed to reveal the typical febrile reaction which is wont to be seen after simple mastoidectomies. The sharp

febrile rise and gradual drop of the temperature curve was absent. Instead there was a slight range of temperature between 100° and 101° for the next few days.

Differential blood counts were made during this period and gave results as follows:

January 11. 18,200 leucocytes; 81 per cent polys, 19 per cent monoes.

January 12. 23,400 leucocytes, 86 per cent polys, 14 per cent monoes.

Headache was still complained of and the child was restless. A lumbar puncture was performed with the following findings:

Physical. Clear fluid, three cells to the c.c.

Chemical. Copper reduction present. Albumin present. Globulin negative.

Cytological. 100 per cent lymphocytes.

Bacterial. Negative to smear and culture.

The heart gave no symptoms except that there were periods of the day when the pulse was reported as bounding. Its rate lay between 100 and 110.

This condition continued until January 13, when, in the afternoon, the temperature rose to 102.4°, dropping on the morning of the 14th to 100.2° and rising again in the afternoon to 103.4°. A blood culture taken at this time was negative. Differential blood count gave 26,400 leucocytes, 78 per cent polynuclear, and 22 per cent mononuclear, leucocytes.

During the next two days the temperature curve developed characteristics of sepsis—lower in the morning and higher in the afternoon, with a gradual elevation of the entire curve, a higher point being reached each afternoon until, on January 18, 1920, it reached 105.2°.

There had, during these days, developed an acute infection in the opposite ear—the left. It was noted that there seemed a markedly reduced faculty for hearing and examination revealed the left membrana tympani to be bulging. This was incised, and a free discharge of pus was established coming away under signs of pressure.

The physical examination now showed the presence of an extra systole, intermittent in character, heard at every third beat. Differential blood count showed 24,000 leucocytes, of which 82 per cent were polynuclear, and 18 per cent mononuclear, leucocytes. Another blood culture was taken and reported sterile.

On January 16, 1920, Dr. Felix Cohn saw the patient in consultation, and all concerned agreed with him to adopt an expectant

attitude in view of the findings in the left ear, which was secondarily infected. In a few days the local findings showed the left ear as clearing, and in few more days the ear was dry, the drum-head normal.

But the febrile state continued, and on January 19, 1920, Dr. Joseph Barsky, the Internist, after careful examination, expressed it as his opinion that the sepsis was of otitic origin and that the heart, although giving signs of disease, was not the cause of the temperature. Differential blood count now showed 15,000 leucocytes, of which 77 per cent were polynuclears and 23 per cent were mononuclears. The urine examination gave a trace of albumin and a few leucocytes.

The patient's condition seemed to be getting worse. The prostration was more marked and the patient complained of pain in his lower extremities when touched or moved. The pulse was very irregular, at times intermittent. The child became drowsy and lay apathetically in one position for long intervals.

A blood culture was taken. At this time it was found sterile; later (after the operation had been performed) the laboratory reported a very few colonies of streptococcus hemolyticus.

It was at this time that the question as to the exact lesion with which we had to deal became the potent factor in the clinical estimation of the case. On the one hand, if the sepsis was due to an endocarditis, then operation not only was useless, but, by its extra drain on the vitality of the patient, added to the gravity of the case. If we were dealing with a septic sinus thrombosis, "time" was a valuable factor, and to accomplish removal of the infected clot and separate the general circulation from the infected area became imperative as soon as there was reasonable belief of such a condition existing. The result of surgery in septic sinus thrombosis is proportionate to the promptitude with which resort is had to surgery. There were two conferences held—at one, Dr. Robert Carlisle and Dr. Isadore Friesner taking part, the patient was pronounced a poor surgical risk and operation was not advised; at the second, with Dr. Joseph Barsky, the patient's father and Dr. Emil Altman, it was recommended to operate and explore the sinus, even in the face of negative blood culture findings. This question will be further discussed below.

It was determined upon to operate, and this was accordingly done on January 19, 1920. At operation pulse was 132, bounding in character, temperature was 103.6° and respirations 26.

The internal jugular vein was exposed and ligated in two places and a section of the vein resected between the ligatures. The neck wound was closed by suture.

The mastoid wound was opened, cleansed of granulations, the sinus brought into view, plugged above the knee and near the bulb, and incised between the plugs. A mural thrombus extending toward the bulb was found and removed. Plugs were applied and the wound packed in the usual manner.

Postoperative period gave temperature elevation from 99.4° to 105.4°, then a rapid remission with a curve between 100° and 102° until January 22. Differential blood count showed a leucocytosis of 15,000, with a differential count of 69 per cent polynuclears and 31 per cent mononuclears. One more blood culture was taken and found positive.

The temperature curve on the 21st was low. On the 22nd there was a rise to 104.8°. Differential blood count at this time showed 20,400 leucocytes, of which 79 per cent were polynuclear, and 21 per cent mononuclear, leucocytes. The curve now began to take larger sweeps. It was not as sharp as before. On January 23, 24 and 25 it remained low, gradually rising on January 26 to 102.6°; then it gently came down on the 27th to normal, where it remained for the remainder of the patient's stay in the hospital.

The wounds progressed satisfactorily and the patient was discharged on February 1, 1920, making a further uneventful recovery.

The heart still presents a systolic murmur; both ears are dry, wounds healed.

COMMENT.

This case presents a number of factors of importance. In the first place, too much stress cannot be laid on the elicitation of the history of the onset of the heart murmur. In the first instance this was noted, but not accorded the importance it warranted. Luckily, the patient's father, a physician, was able positively to place the onset of the heart murmur and the cardiac trouble with the attack of diphtheria some years previously. It was the easier to gauge the fact that the cardiac condition was not radically changed from that in which he had found it at various intervals of time when he had examined his son's heart. This greatly aided in the final determination which resulted in resort to surgery on the internal jugular vein and the lateral sinus.

The so-called painless type of mastoiditis which this patient presented is not the type generally found in children. It is a type

which Boeninghaus, Phillips and others, as well as the writer, have, on occasion, described. This type of case is not accorded the recognition its gravity warrants. It always presents a potential danger because its painless character, free flow of pus from the incised ear-drum and low temperature range are misleading, and give a sense of security as contrasted to the usual cases where spontaneous pain, pain on pressure over the mastoidal area and temperature reactions keep the medical attendants on their guard. The case was recognized and typed from the start. The pathological finding of the hemorrhagic type of mastoiditis accompanied by a perisinus abscess warned of the possibility of an infected sinus.

The limitation of the first operation to cleansing the mastoid of its infection finds justification in the large experience of most otological surgeons that the granulations found upon an exposed lateral sinus in cases of perisinus abscesses are protective attempts of nature to place barriers across the path of invasion, and in the majority of instances where such granulations are left in situ undisturbed, healing and repair rapidly ensue.

The patient failed to give the wonted febrile reaction after the first operation on the mastoid. Since Harris, in his studies of postoperative temperatures, published his findings, we have almost always seen the cases which eventually do well and progress normally to healing and recovery show a sharp febrile reaction within twelve to forty-eight hours after operation. In the majority of instances where such febrile reactions are absent, especially in children, the writer's experience leads to the expectation of some untoward finding. This observation has had substantiation in this case also.

Finally, the situation which developed postoperatively, when the question arose whether the sepsis was due to a lighting up of the old endocarditis from the infected mastoid process or whether there was a septic sinus thrombosis present, was a serious and momentous one. In the report of the case we have cited the situation as it presented itself. Here only a few additional facts need mention. The data for the diagnosis of either the one or the other condition was not complete before operation, for repeated blood cultures had been reported negative. Even had the report been positive, the issue would still have been a mooted question, for a positive blood culture could conceivably be a factor in either septic endocarditis or septic sinus thrombosis. In sinus thrombosis, as a rule the leucocytosis is higher than in septic endocarditis, but upon this laboratory aid too much stress cannot be put. Careful bedside studies of the behavior of the heart, the fact that it seemed adequately

compensated, and furthermore, that it showed by every means of diagnosis at our disposition no evidence of change as to bulk or position—these facts added to the local finding of a perisinus abscess at the first operation settled the question for the writer. For others that studied the case the decision was arrived at by empirical reasoning: Septic endocarditis meant a fatal termination, the child was rapidly getting worse and the exploration of the sinus and the localization of the septic foci in the blood vessels offered the only chance for a recovery. The events justified the chances taken.

In this connection it must be noted that in the terminal stages of septic sinus thrombosis a septic endocarditis is very often one of the lesions presented. Here the acuity of observation of those having charge of the patient governs; and where, as here happened, the child was under competent observation from the onset of its illness, confidence in the findings made at the various physical examinations excluded the idea that the heart was the seat of an endocarditis secondary to the septic sinus thrombosis.

In conclusion, it must again be stressed that, even in the face of repeated negative blood culture findings, the diagnosis of septic sinus thrombosis may be made upon the clinical findings alone. Such was the situation in the case here described. The findings were reported positive after the operation was finished and a clot had been demonstrated. There was one more positive culture after operation, and thereafter, the sepsis subsiding, no further blood examinations were made.

Case III. Extensive Sinus Thrombosis Extending Backward Beyond Knee of Lateral Sinus—Atypical Temperature—Positive Blood Culture—Operation—Bilateral Facial Edema—Recovery.*—R. S., female, aged 6 years, came under observation February 1, 1920, having an acute otitis media with symptoms of mastoidal involvement which followed an attack of influenza. There were the usual local and systemic symptoms—pain and tenderness over the mastoid process, reduced hearing, persistent temperature and a bulging membrana tympani on the left side. Paracentesis was promptly performed and a discharge from the ear established. The infecting organism was a streptococcus.

The condition continued unabated in spite of local and systemic treatment, and at the end of a week, because of this and increasing prostration, the child was admitted to the Stern's Private Hospital.

The simple mastoid operation was performed February 9, 1920. The lesion in the mastoid process was of mixed type—in parts there

*Patient was presented, Otological Section, New York Academy of Medicine, March, 1920.

was evidence of a coalescing process, and in parts there was evidence of a hemorrhagic type of infection. Frank pus was found in most sections. The bony sinus wall covering the lateral sinus was found diseased and was removed. The sinus wall was normal to macroscopic appearance. The wound was packed in the usual manner and the skin-wound partly sutured.

The postoperative period showed the usual rise in temperature, the curve reaching 102.8° on the day following operation, then dropping on the second day and remaining low, ranging about 100°. Pulse was 108 to 110 and respirations 20 to 22.

The child seemed to be progressing satisfactorily, although there was continuously complaint of cephalalgia. At change of dressings nothing unusual was to be noted.

On February 17, seven days after operation, the patient became very restless and was drowsy most of the day. The temperature, which began rising on the evening of the previous day, continued to mount during the night and reached 105.8° in the afternoon. Pulse was 124 and respirations 26.

In view of the findings at operation, which had shown an involvement of the bony wall covering the lateral sinus, the suspicion of sigmoid sinus thrombosis was at once suggested, even though no data to substantiate such a diagnosis was at hand. A blood culture was negative. The blood showed 30,200 leucocytes, of which 80 per cent were polynuclear, 17 per cent small mononuclear, and 3 per cent large mononuclear, leucocytes. The hemoglobin index was 77 per cent (Sahli); color index, one minus; there were no nucleated reds, anisocytosis, poikilocytosis nor polychromatophilia.

A general physical examination revealed no abnormality in heart and lungs or in the abdominal organs. The temperature on February 18 dropped to 103° and ranged about 103° to 104.8°. Clinically the patient showed no change. There was considerable complaint of pain in the head, rather indefinitely localized over the left side, and there were some indefinite pains in the throat. Examination was, however, negative. A blood examination on this day showed a lessened leucocytosis; namely, 20,000 leucocytes, but the polynuclear leucocytosis was slightly increased—it was 84 per cent, with 13 per cent small, and 3 per cent large, lymphocytes. The possibility of a pneumonia was entertained, but no physical signs justified that diagnosis, although there was an increase in the respiratory rate to about 30, the pulse remaining about 118 to 120.

February 19, 1920, showed the condition unchanged. Pain was still complained of on the right side of the head and neck. Exam-

ination was still negative in every respect. The temperature rose on the afternoon of this day from 101° to 104.4°. The blood examination showed 18,600 leucocytes, of which 86 per cent were polynuclears and 14 per cent mononuclears. The child seemed more drowsy, and because of this and the cephalalgia, although all other signs were negative from a neurological standpoint, a lumbar puncture was performed and 12 c.c. of clear fluid removed. It came away under slightly increased pressure, but was normal physically, chemically, cytologically and was sterile to smear and culture.

On February 21 a normal temperature was exhibited in the morning, but at noon the child developed a violent chill which lasted twenty minutes, and the temperature rose to 105°. During the succeeding hours the temperature gradually fell to 98°. The general appearance of the child was poor. There was marked prostration and exhaustion, and the tongue and general condition denoted sepsis.

Another blood culture was taken and reported sterile. After the exploration of the lateral sinus and the demonstration of the extensive clot, Dr. Lester Unger, the pathologist, made subsequent report of finding streptococcus hemolyticus in this culture.

A consultation with Dr. Joseph Barsky, the Internist, was had, and it was determined to explore the lateral sinus upon the clinical findings, in the absence of any signs denoting a focus of infection elsewhere. It was decided upon not to await the outcome of the blood culture in view of the gravity of the patient's general condition.

The neck was operated upon first. The internal jugular was exposed and a section of the vein resected, and the wound closed by primary suture. The vein specimen seemed normal with fluid contents.

The mastoid wound was then opened, granulations removed, and the lateral sinus exposed from the bulb to the knee. A fully organized obliterating thrombus was found, extending into the bulb. The bulb was cleared as far as it was possible to reach, and then a drain introduced. The incision was extended backward toward the torcular end of the sinus about one and one-half inches before free bleeding was obtained. Then this end was plugged off. The wound was packed and the usual dressings applied.

The patient rallied from the operation under appropriate stimulations and supporting therapy, and seemed more comfortable, except for a chill lasting about twenty minutes during the first night. The next morning the general condition was more satisfactory.

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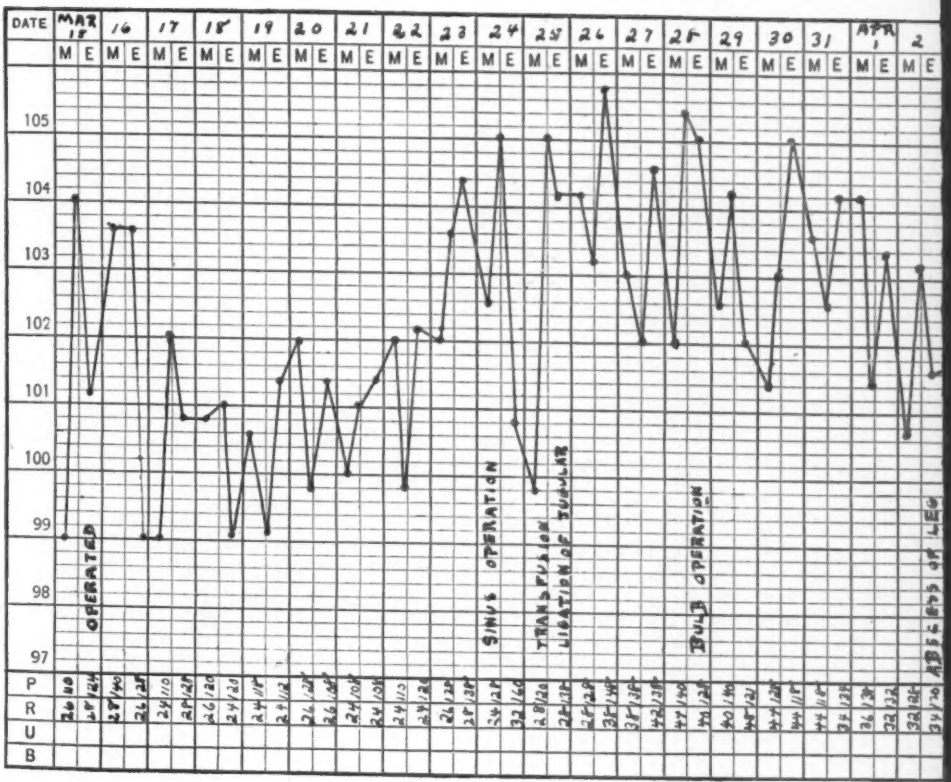
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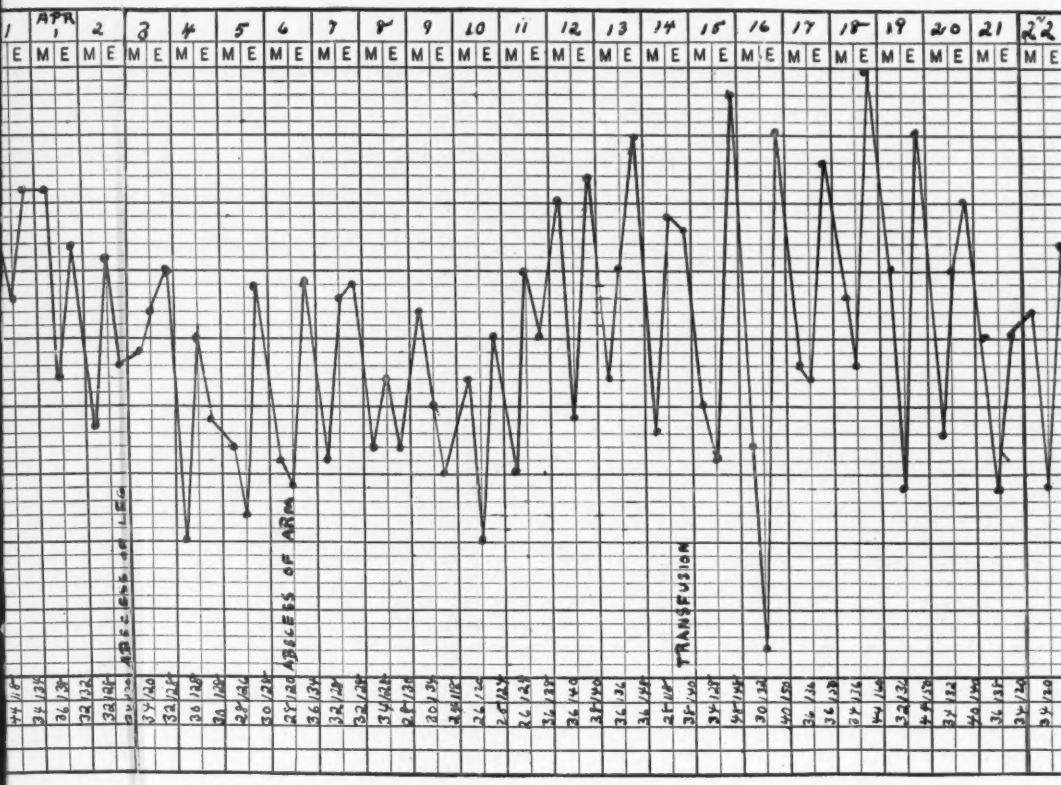
Case VI. Contracted Chart of R. K.—Septic Sinus Thrombosis — Otitic Sepsis. Dr. Kopetzky.



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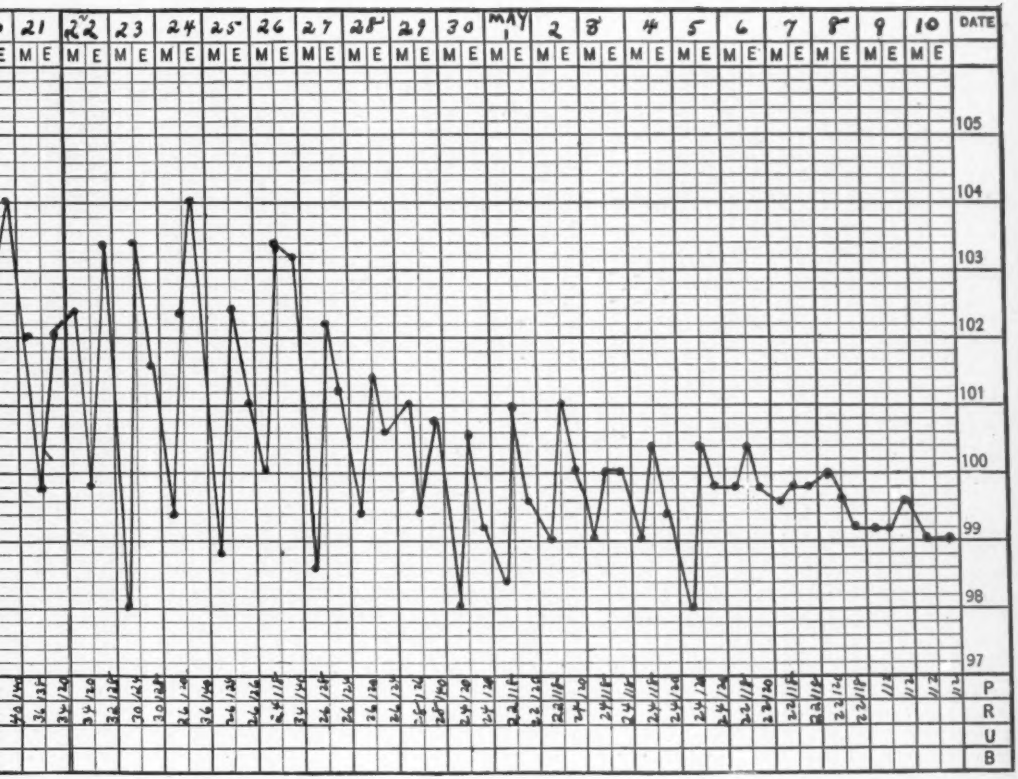
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On February 23, 1920, the temperature remained between 100° to 101°, pulse 104 to 120, respirations 22 to 26.

On February 24, 1920, there appeared a peculiar swelling over the parotid area of the left side, extending upward to the root of the zygomatic process and forward to the left angle of the mouth and downward under the ramus of the jaw to the neck. The lower eyelid on the left side was included in the edematous mass. The skin over this area was not inflamed, was freely movable, and in no way gave the impression of acute inflammation. It looked and felt like an edema. There was no evidence of glandular enlargements; no outline of gland masses was palpable. There was difficulty in swallowing and respiration due to the same form of edematous swelling pressing upon the pillars of the left tonsil and the posterior-lateral pharyngeal wall, but the tonsillar fossa showed no abnormality, nor did the uvula show any edema. On the area about both the mastoid and the neck wound, there was no evidence of either redness or other signs of infection. The temperature rose to between 102° to 103°, pulse 112 to 116, respirations 26.

It was thought at first that an erysipelatous infection was the cause of this condition, but the absence of any discoloration of the skin overlying the edematous area, the absence of a line of demarcation, the absence of septic temperature and the presence of swelling on the pharyngeal wall discredited such a diagnosis. No treatment was administered as the child's general condition continued good, the temperature dropping on the next day to 100° to 101°, pulse 120, respirations 26. Nevertheless, the swelling grew more pronounced, extending outward. Its margins were not extended nor sharply defined. The eyelid became free, but the neck area below the chin and ramus of the jaw grew larger. It seemed as if the mass dropped slightly downward by gravity. The bed was then so arranged *that the feet were elevated and the head lowered.* On the following day a decided improvement in the size of the swelling was noticeable, swallowing and respiration were easier, but a similar condition, identical in all respects, developed on the opposite side (right). It began, *not* in contiguity with the swelling on the left side, but just anterior to the ear lobe, rapidly involved the right side of the face and neck below the ramus of the jaw, and extended downward on the neck. Coincidentally the scalp behind the right ear became edematous. Here, too, absence of redness, lines of demarcation or outline of gland masses was noted. The picture presented by the child's face was that of an exaggerated picture of

mumps, the whole face, chin and neck on both sides being much enlarged.

Meanwhile the general physical condition of the patient remained good. There were no further chills, the temperature, except on the day of dressing, when it rose after the change of drain-plugs in the sinus to 103°, remained within comfortable bounds, between 99° and 100°, and the status of convalescence set in, except for the bilateral facial and neck swelling.

The swelling lasted about two weeks. It began gradually to subside at the end of nine days and was gone at the end of fourteen days. The swelling on the right side was the last to disappear. During the entire period the pulse, respirations and temperature were practically normal and the wounds progressed favorably. The neck wound was healed, stitches removed, and the mastoid wound was granulating favorably.

During the period when the swelling was at its height, various consultants saw the patient, among them Drs. Murray Bass, Harold Hays and Emil Altman, as well as the family physician, Dr. Benjamin Kaufman; and a decided difference of opinion prevailed as to the diagnosis and etiology of the condition presented. All agreed that we were not dealing with an erysipelatous infection. Some held that it was a case of mumps. Against this, the location of the swelling, the absence of pain, the lack of temperature, the long duration of the lesion and the failure to show intensity of inflammation over the area of the parotids were noted. The diagnosis of angioneurotic edema was also advanced. Against this were the absence of any irritability of the area, the single onset, the absence of any neurotic signs, the age of the patient and the improvement when the feet of the bed were elevated.

The writer held that it was a case of edema of unknown causation, probably due to a derangement of the superficial circulation in the venous channels in the effort of nature to establish collateral circulation. This explanation is, of course, also open to a critique, but it seemed at least to offer some explanation for the unusual phenomena. It is presented here as a matter of record, as a finding which the writer has never before observed in this type of case.

From March 9, 1920, convalescence continued, the patient being discharged from the hospital thirty-three days after admission.

Examination on July 1, 1920, showed all wounds healed, and the child fully recovered.

COMMENT.

This case presents several interesting features. In the first place, the case was under the writer's observation from its onset. There

had been the debilitating influence in the etiological factor, namely, the influenza, and no time was lost in the performance of the primary mastoid operation. In the second place, at the primary operation the bony covering of the lateral sinus was found diseased and removed, and the wall of the blood vessel presented a normal macroscopic appearance.

The usual postoperative temperature curve is presented, and then an interval of a week intervenes without symptoms of moment. Following this, there ensues a gradual elevation of the temperature, and cephalalgia. There is nothing characteristic of septic sinus thrombosis in the character of the fever chart, and the blood cultures are negative. The differential counts are not specifically significant.

At this time, there is absolutely no sign pointing to the sinus as the seat of an infected focus, nor any indication to justify surgical intervention on the sinus. Yet a thrombus is developing, for, five days later, when a distinct chill prompts intervention, a thrombus is found, so extensive as to be almost ineradicable. The late finding of a positive blood culture is to be noted. Had exploration of the sinus been postponed until the report of a positive blood culture had been obtained, then operative interference would have been, in all probability, too late. One is justified in stressing the contention that, where the primary focus of the infection is found at operation to reach to and include the sinus at the primary mastoidectomy, then, when untoward symptoms supervene subsequently, unaccounted for by lesions in any other portion of the body, exploration of the sinus is indicated to locate and eventually eradicate the infected blood vessel. At such secondary explorations of the sinus, the internal jugular should be resected or ligated before the secondary surgery on the lateral sinus is undertaken.

Regarding the facial and cervical edema which supervened subsequent to ligation of the internal jugular on the affected side, one can only speculate. There are no proofs as to just what occurred. Edema of the dura and brain tissues have been reported, but as far as the writer is aware, he has never observed nor seen reported an edema located as this one was. The observation is here placed on record, and the fact that placing the bed so that the head was lower than the feet seemed not only to give the patient the most comfort, but also to relieve the situation. Further study and reports of such findings would be welcome so that the condition might be the better comprehended.

Finally, one may view with equanimity the temperature rise which follows the replacement of the drainage plugs in the sinus lumen during the first few changes of dressings after operation. A certain amount of infectious material is freed by the procedure, and this is signalized by an elevation of temperature.

Case IV. Primary Jugular Bulb Thrombosis, Presenting Symptoms Suggesting Extension Toward the Cavernous Sinus—Streptococcus Viridans in Blood—Operation—Recovery.*—On March 23, 1920, F. B., aged 5 years, was admitted to Beth Israel Hospital.

His illness came on rather gradually, beginning with a period of hoarseness and general malaise about ten days previously. There were cough and headache which became noticeable seven days before admission. Three days later the temperature rose to 103°, but soon dropped.

During this period the child was treated expectantly by Dr. Nicol M. Mandl, the lungs being suspected as the probable seat of the lesion, although it was conceded that no definite physical signs had as yet become determinable.

The ear was first brought into notice the evening before admission when pain in it was the chief complaint, accompanied by a rise in temperature to 106°.

During the morning of the day of admission, after a spell of coughing, the patient spat up some blood. The pain in the right ear persisted, and the case came under otological supervision. Examination showed a bulging membrana tympani which was immediately incised, and drainage of pus established from the tympanic cavity.

At this time the child looked acutely ill, face flushed, temperature 104.6°, pulse 140 and respirations 38. The eyelids were swollen, more markedly in the right eye, and the conjunctivae, particularly of the right eye, were injected and edematous. There was a noticeable rigidity of the neck.

Admission to the hospital was advised for further observation and, eventually, operation, as the gravity of the situation was already comprehended.

The details of the examination upon admission are as follows:

Eyes. The conjunctivae are injected and there is a scleral injection over the upper and outer part of the right eye, with a discernible puffiness of the upper eyelids, the right eye being more markedly edematous than the left. The eyegrounds are normal.

*Patient was presented, Otological Section, New York Academy of Medicine, March, 1920.

Ears. There is a purulent discharge from the right ear and the mastoid process on the right side is moderately tender to pressure. The left ear is normal.

Lungs. There is a slight dulness over the right base; otherwise percussion is normal. Some inconstant subcrepitant rales are present over right base posteriorly.

Reflexes. On the whole, normal. There is no Kernig nor Babinski present. A hyperaesthesia of both lower extremities is noted. There is a rigidity of the neck to flexion.

The *throat* is congested, with no indurations of the tonsillar pillars or pharyngeal walls. *Heart* normal.

Abdomen. No abnormality noted.

The pus from the ear discharge presented streptococci on smear.

Examination of the blood showed 4,000,000 red cells, 19,200 white cells, hemoglobin index 65 per cent. Differential count gave 84 per cent polynuclear leucocytes and 16 per cent small lymphocytes.

Blood culture was taken, subsequently to be reported as positive.

A *lumbar puncture* showed the following in the cerebrospinal fluid:

Physical. Tinged with blood.*

Cytological. 260 cells to the c.c., 60 per cent polynuclear and 40 per cent mononuclear.

Chemical. Copper reduction present. Albumin present.

Bacterial. Sterile to smear and culture.

Urinary Analysis. Negative, except for heavy trace of albumin.

Radiograms. (a) Of the *chest,* showed increase in the hilus and lung markings and numerous calcified foci. Lung markings indeterminate; (b) of the *mastoid process,* showed increased density on the right side, but no evidence of cellular destruction.

While it was recognized that the case was grave, yet, in the absence of the final report on the blood culture, the absence of classical signs of mastoiditis, and in view of the short time of the acute stage of the illness, it was deemed expedient to await further developments, particularly as the medical attendants could not yet rule out of consideration the question of lung involvement.

The next day gave no improvement in the condition of the patient. On the contrary, the rigidity of the neck was more pronounced. There was a Kernig becoming evident. The edema of the eyelids and the congestion of the conjunctivæ were more pronounced.

*Because of the contamination with blood, no reliance could be placed upon the cytology and chemistry of the fluid at this examination.

A second blood examination gave 28,000 leucocytes, of which 86 per cent were polynuclears and 14 per cent mononuclears. On the morning of this day the temperature had dropped to 99.8°, pulse 128 and respirations 34, to be followed in the afternoon by another abrupt elevation, to 104.4°. Cephalalgia and the purulent discharge from the ear were the predominating symptoms, accompanied by the septic temperature curve.

The medical attendants felt that the symptoms from the chest were negligible—the cough had disappeared—and expressed it as their opinion that the clinical manifestations of sepsis, prostration and cephalalgia had no origin in any pulmonic lesion.

The report from the blood culture was then at hand, and the positive bacteremia was established to be due to streptococcus viridans in the blood. Dr. L. G. Hadjopoulos, bacteriologist to the hospital, transplanted and positively identified the strain.

The local signs of acute mastoiditis were still absent. There was no drooping of the posterior meatal wall, no edema of the periosteal covering of the mastoid process, nor were there definite points of tenderness over the classical landmarks to warrant a diagnosis of mastoiditis. The radiograms of the mastoid were also inconclusive insofar as they failed to show bony cell wall destruction, although they did denote increased density on the affected side. Such a finding would have been the usual radiographic picture of any acute otitides of ten days' duration. No reliable data upon which to fix indications for operation was therefore available from these sources.

On the other hand, when it was considered that the patient presented a sepsis accompanied by increasing general prostration, that the cephalalgia became more intense and that there were presented advancing edema of the eyelids and conjunctivæ and neurological findings suggestive of an intracranial focus of infection —all this in addition to a purulent otitis with a positive blood culture — then, in view of the fact that no other origin was determinable as a cause of the clinical picture, the diagnosis of primary bulb thrombosis was made. It was thought that, because of the eye symptoms and the rigidity of the neck, the petrosals were probably involved in the pathologic lesion. Operation was therefore urgently recommended.

The operation was accordingly performed late in the afternoon of March 25, the time of operation synchronizing with a drop in the patient's temperature.

The internal jugular vein was first separated and ligated. The Boston incision was employed in reaching the vein.

Then followed the performance of the simple mastoidectomy. The mastoid infection uncovered was of the hemorrhagic type. There was hardly any destruction of bony intercellular walls. Some frank pus was observed in the tip cells and in the cells adjacent to the sigmoid sinus. No frank pus was found in the cells adjacent to the mastoid antrum. The sinus was freely exposed. The vein wall was normal in appearance. No clot was demonstrable. Drains were placed in the bulb; the upper sinus end was plugged and the wound packed in the usual manner. During the operation 250 c.c. of saline were given by hypodermoclysis.

Immediately following the operation, improvement was noted. The temperature dropped to 100.4°, respirations 36, pulse 128.

The next day the height of the temperature was 102.8°, gradually dropping to 100°. It rose again to 102°. The pulse ranged between 123 and 128; respirations were about 34. Wound pains and headaches were still complained of, and some pain was experienced during urination.

A postoperative differential blood count showed 28,000 leucocytes, of which 74 per cent were polynuclears and 26 per cent small lymphocytes.

On March 27 the highest temperature range was 101.6°, but during most of the day it ranged between 99° and 100°. The child seemed out of danger, rested well and slept during the greater part of the night. Pulse was 104 and respirations 28.

The following day the afternoon temperature rise was 100.4°. During the day the range was between 99° and 100°. Pulse 98 and respirations 28. The edema of the eyelids was disappearing The neck was flaccid; the child was relaxed.

During the afternoon there became noticeable a swelling on the *left* side of the neck extending upward toward the parotid gland, which it seemed to encompass. This began to disappear ten days later, and in a few days more was gone. Temperature ranged between 99.4° and normal. Pulse 98; respirations 28. Blood examination showed a total count of 13,600, with a differential of 75 per cent polynuclear, and 25 per cent mononuclear, leucocytes.

Thereafter convalescence was uneventful. The neck wound healed by primary union; the mastoid wound was granulating satisfactorily. A blood culture subsequent to operation was reported sterile.

The patient was discharged April 3, 1920, and was subsequently dressed until the mastoid wound had entirely healed.

The child was examined July, 1920, and found fully recovered.

COMMENT.

Primary jugular bulb thrombosis probably occurs more often than it is recognized. This is particularly true among the younger patients—the infants and children. The difficulty lies in convincing the medical attendants of these patients that *the jugular bulb and the sinus may become the seat of an infectious process without there being a mastoiditis as an intermediate lesion.* When McKernon in 1904 (Transactions of the American Laryngological, Rhinological and Otological Society) first called the attention of the American profession to this lesion and its clinical picture, he stressed this same point.

Much valuable time is usually lost while efforts are directed to eliminating other possible factors and elements as the cause of the symptomatology which the patients exhibit. To be successful, resort to surgery on the internal jugular vein must be very promptly taken.

Nothing is herein contended which should be construed to mean that proper and thorough general physical examinations and the data they present should not receive careful consideration, but too long a delay while expectantly waiting the development of a probable clinical picture whose existence is conjectural should not be used to ward off surgical intervention. In the interest of saving life, exploratory procedures offer, in obscure cases, a better chance of saving life than an operation so long delayed that meningeal or other secondary septic foci are presented at the time of operation. In this connection, Case V offers instructive data.

With the newer means of laboratory aids to diagnosis now at our disposition, the diagnosis should be made promptly. The factors in determining the diagnosis are few. *A sepsis unaccounted for by any other demonstrable lesion, a purulent otitis media and a positive blood culture constitute the principal elements.*

When the anatomical relationship between the dome of the jugular bulb and the floor of the tympanic cavity are recalled, their intimate contact must be appreciated. In addition, in about 20 per cent of the cases among children (personally, the writer has seen it eight times in twenty-five specimens) there are dehiscences in the bony tympanic floor, leaving the mucous membrane of the middle ear directly in contact with the wall of the dome of the jugular bulb. From this, the mechanics of invasion of the jugular bulb are comprehended.

In many of the cases recognized early enough and operated upon, the involvement of the mastoid process is negligible.

In Case I of this series, the primary thrombosis of the jugular bulb was entertained as the diagnosis by both the medical attendants and

the writer, but because of the absence of a positive blood culture it was for a time held in abeyance. In the case here discussed (Case IV), symptoms from the lung were, for a time, the misleading element. Fortunately, the diagnostic data were complete, viz., purulent otitis, sepsis and positive blood culture; and the diagnosis was conceded and operation permitted early enough to save life.

While it is not held that the edema of the eyelids on the affected side, accompanied by a conjunctivitis, an increasing rigidity of the neck and a Kernig, are sufficient in themselves to establish a diagnosis of petrosal involvement toward the cavernous sinus, yet they are danger signals which warrant recognition as probably meaning such. In other cases coming under the writer's observation later in their course, the fatal termination supplied proof, happily lacking in this case.

Primary jugular bulb thrombosis may occur accompanying a mastoiditis which presents classical symptoms. Here the danger lies in misinterpreting the entire clinical picture. The sepsis may be ascribed to the mastoiditis. In the light of our experience, mastoiditis does not give a sepsis early in its course, nor should the finding of a positive blood culture early in the course of the disease permit limiting the surgery to the mastoid process. In such a contingency, exploration of the sinus and jugular bulb are indicated in addition to the surgery on the mastoid process, although the procedures may be done in successive stages, an interval of time elapsing between the operation on the mastoid and that on the blood vessels.

Finally the absence of mastoiditis in no way must be held to inhibit a diagnosis of primary bulb thrombosis or to contraindicate operation to eliminate the lesion in the bulb.

Case V. Primary Jugular Bulb Thrombosis in Painless Type of Mastoiditis—Sepsis with Absence of Symptoms of Mastoiditis—Positive Blood Culture—Determination of Side Involved—Operations—Unusual Neurological Findings—Death.—S. K., female, aged 6 years, was admitted to the Park Hospital on May 11, 1920.

Having recovered from an attack of measles three weeks previously, the child began to complain of pain in her right ear, soon followed by involvement in the left ear also. Five days later, both eardrums were incised and local treatment instituted. The ears discharged very profusely for about two weeks, when the discharge stopped in the left ear. During this time the temperature elevations were already high, and the left ear was again incised; but no discharge of pus was obtained.

One week before admission, with temperature elevations as high as 103° to 104°, both ears were again incised because of absence of a discharge from both middle ears. There was no complaint of pain locally.

The temperature continued, ranging up to 105, until three days before admission, when severe persistent headache was the chief complaint. The otologist in attendance again incised the eardrums and a discharge was obtained which lasted about twenty-four hours, when the ears were again dry.

The temperature and cephalalgia continued, and some edema developed about the right ear. A radiogram was taken of the mastoid processes and pronounced negative.

The writer saw the child that day and found a septic patient, with chilly sensations but no pronounced chills, a temperature of 105°, a slight discharge from both middle ears, a beginning Kernig and some rigidity of the neck. At this time no other abnormality was noted. The mastoids were devoid of classical symptoms.

Cultures were at once taken from the ear discharge, a blood cell count was made and a blood culture taken.

A tentative diagnosis of sinus thrombosis was made. It was extremely difficult to convince the medical attendant and the pediatrician who had been called into consultation as to the involvement of the ear. The absence of the discharge during the high range of temperature failed to be accorded its due significance. The sepsis had until then been unaccounted for, and an expectant attitude adopted awaiting a more definite development of a clinical picture. In the absence of laboratory aids to diagnosis, which, up to the time of the writer's visits, had not been used, no further data were available. The tentative diagnosis was therefore based on the history of the case and its clinical picture, and was arrived at by a process of elimination of any other sources of the sepsis.

The culture from the discharge from the ears gave pure culture of pneumococcus.

The preliminary report of the blood culture was negative; the final report gave the streptococcus hemolyticus. The blood-cell-count was as follows: 39,000 leucocytes, of which 78 per cent were polynuclear leucocytes, 20 per cent small, and 2 per cent large, lymphocytes. Hemoglobin 80 per cent (Sahli). Red cells, 4,200,000. Color index 0.95. No nucleated red cells.

Urine. Turbid; in color, amber; acid in reaction. Heavy traces of albumin, no sugar, heavy trace of acetone but very faint trace of diacetic acid. Microscopically, there were a few leucocytes and some pus clumps. No casts nor crystals.

The diagnosis was then conceded and the child admitted to the hospital for operation. Temperature 103.4°, pulse 124 and respirations 22.

The physical examination at admission added nothing to the data already available except that the heart appeared enlarged. Everything else was negative. Both ears were discharging moderately, and the discharge was not coming through the drum membrane under pressure-pulsations. There was no drooping of the posterior superior canal walls, nor was there any tenderness upon ordinary pressure over the mastoid process. Upon deep and strong pressure the right mastoid showed some tenderness. The patient was septic in appearance and apathetic.

The question that then presented itself was the determination of the side upon which to resect the jugular vein. It was not possible by any means at our disposition to differentiate the sides prior to exploration of the mastoid processes. The eyegrounds gave no help. Palpation gave nothing and the most painstaking examination of the ears failed to give any data of value. The radiograms of the mastoids were of no help.

It was decided to explore both mastoid processes; and, in the event of obtaining data upon which to differentiate between them, thus try to locate the side upon which there was a thrombus.

This was accordingly done. The right mastoid showed no bony destruction and hardly any involvement. The left mastoid presented the usual hemorrhagic type of infection, some purulent granulations. The sinus wall was therefore exposed upon the left side.

The exposure showed a discolored sinus wall which, when compressed, did not fill up promptly with blood. Plugs were placed and the sinus opened. A very sluggish bleeding followed when the sinus was plugged above and the bulbar end remained unplugged. When plugged below and the upper plug removed, free bleeding ensued.

The upper plugs were replaced and the incision carried into the entrance of the bulb, and as much clot as possible removed. Drains were applied and the wound dressed in the usual manner. Ligation and resection of a section of the internal jugular on the left side followed.

Postoperatively, the patient reacted badly. The temperature dropped to subnormal, 97°, the pulse 88 and the respirations 18. Appropriate stimulants were given but the patient remained in a condition of shock and collapse during the next two days. There were no other symptoms. Nourishment was given, and salines administered, by proctoclysis.

On May 14 reaction set in. The temperature rose to 104.8°, and between May 14 and May 17 assumed a typically septic curve, rising in the afternoon and dropping to normal in the morning. The pulse ranged between 82 and 120 and was frequently weak and irregular; respirations were between 20 and 24. During this period there were attacks of vomiting, and although nourishment was taken, it was often refused or vomited soon after ingestion.

On May 17 a second blood culture was taken and showed many colonies of streptococcus hemolyticus. Because of the increasing cephalalgia and the more marked rigidity of the neck, a lumbar puncture was made for examination of the cerebrospinal fluid. It resulted as follows:

The fluid was under increased tension.

Physical. Clear.

Chemical. No abnormal constituents.

Bacterial. Negative to smear and culture.

Cytological. Four cells to the c.c.

Both eyes now became swollen. The conjunctivæ were inflamed and some secretion was formed under the lids. Local, cold applications and treatment soon caused a subsidence of this symptom.

The child seemed unimproved although the temperature was lower—between 101° and 102.8°—but the pulse was weaker and often irregular. On May 18 she became incontinent, vomited frequently and was so drowsy as to be aroused with difficulty.

In view of the septic condition, which seemed uncontrolled, and to give the patient a better fighting chance, a blood transfusion was given by Dr. Lester Unger, the father of the patient being the donor. 350 c.c. of blood were introduced.

On May 20 the child was weaker, thirsty and drowsy, with involuntary urination. The patient experienced difficulty in swallowing. Following the transfusion of blood, the heart was dilated and the apex-beat was found outside of the left nipple line. The sounds were of fair quality. The pulse was small and easily compressible.

A sharply outlined swelling is apparent on the right side of the neck high up under the angle of the jaw. A positive Kernig is present. The temperature is again high, 104.5°.

The entire appearance of the patient is changed. She lies in a semirecumbent position, markedly somnolent, legs drawn up on the abdomen, with the right arm completely relaxed. She does not seem able to make any voluntary movement with the right arm, not even in response to stimuli. The arm offers no resistance to movement

and the patient seems to have no power to move it. The other extremities are normal. There is apparently an anesthesia of the right hand and right forearm, and the superficial reflexes below the shoulder are absent on the right side. Mentally, the patient responds slowly to simple commands. Kernigs are markedly present. There is no Babinski nor ankle clonus. Sensation is normal in the lower extremities. The abdomen is slightly sunken but the viscera are apparently normal.

The heart gives a loud, presystolic murmur; the pulse is easily compressible.

The neck is rigid; the cervical glands at the angle of the jaw on the right side are swollen and tender. Respirations, regular and deep. No facial paralysis, and deglutition uninterfered with. Temperature ranges between 102° to 104.6°; pulse 92 to 120; respirations 24 to 30.

On May 21 the patient's condition became critical and convulsions set in, becoming more frequent and more intense throughout the day and part of the night. They occurred about every ten or fifteen minutes. The temperature ranged between 101.8° to 103.6°; the rapidity of the pulse reached 160, and it was weak and irregular.

On May 21 sensation and movement returned to the right arm. The patient voluntarily moved same. The mental state, too, was improved. The patient retained food, and asked for it.

An examination of the cerebrospinal fluid at this time showed the presence of numerous pneumococci in it, and during the ensuing week the patient rapidly developed the classical picture of meningitis.

For the purposes of this paper, the details of the remainder of the case-history present nothing of moment. Briefly outlined, we find that at first there was a period of improvement with only an occasional convulsion. There was a complete return to normal of motility and sensation in the right arm and leg; the enlargement of the heart disappeared and the murmur was lost. The temperature ranged high, between 101° to 104.8°.

On May 24 a lumbar puncture was done, 20 c.c. of cloudy fluid being withdrawn under moderately increased pressure. The cell count was 540, and a smear showed an encapsulated diplococcus. Culture showed a mixed infection of pneumococci and short chain streptococci.

Toward the end of the week the signs of meningeal irritation increased rapidly—the patient was irritable and restless, and became involuntary. She was unable to sleep and screamed frequently in

agony. She vomited often and refused nourishment. The limbs became spastic and the neck rigid.

From May 28 until June 3, when the patient died, the meningeal symptoms rapidly became aggravated, pulmonary edema and a left hemoplegia developing on June 2; and a terminal temperature of 109° developed a few hours before the end.

COMMENT.

Particularly among pediatricians, and to some extent among general practitioners also, it has become customary not only to study the ear conditions presented by their patients in acute systemic infections, but to decide for themselves when incision of the membrana tympani is indicated. Additionally, many of these perform their operations themselves, upon the results of their own judgment of the indications. A recognition of the rarer forms of middle ear infections and of atypical cases is hardly to be expected of men whose life-work lies in other fields. Fortunately, the self-confidence of such men in their estimation of the indications for the so-called paracentesis of the membrana tympani is justified by a knowledge of the simpler and more common middle ear infections. In many instances, on the other hand, the little patients become the victims of mistaken zeal with the paracentesis knife.

Entirely aside from the facts adduced by Case V, the little operation of cutting into the drumhead requires as nicely a balanced judgment as does any other surgical procedure. That many ears are incised without sufficient indications became evident to the writer from the harvest of destroyed drumheads coming under his observation which resulted from such promiscuous surgery on the membrana tympani during the recent influenza epidemic in New York City. Because, during an influenza epidemic, a patient shows temperature and a reddened drumhead—but no other symptoms (in some cases no time was allowed for symptoms to develop)—constitutes no adequate indication for incising the drumhead. Cases were seen in which repeated incisions of the same drumhead, as many as four in twice as many days, produced no purulent middle ear discharge. It seems as if the incision is made and remade until a middle ear, which may be reddened and yet contain no pus, is to be forced to disgorge its hidden purulent contents.

What actually happens in many instances, is that eventually an infection supervenes from the outside upon the traumatized drumhead, and the middle ear becomes infected *after* one or other of the repeated incisions of the membrana tympani.

The subsequent life story of these victims, who come under otological supervision later in their lives with middle ear adhesions,

multiple scars or permanent perforations together with a consequent loss of hearing acuity, remains unknown to those whose practices are here criticised. Or, the secondary infection referred to above supervening, otitides eventually develop upon a body deteriorated by some other condition, and thus some quota of the graver cases met with is accounted for.

Incision of the membrana tympani should be *promptly* done to *evacuate pus* in the tympanic cavity, and for no other reason. When pus is present in the tympanic cavity and the incision has been properly performed, then, the pus within the tympanic cavity being, by the nature of the disease and the anatomy of the parts involved, under considerable pressure, such an incision will not close as long as there remains pus under pressure forcing it outward. One cannot pause here to discuss this item in fuller detail. Suffice it to call attention to this factor as elicited by the case-history above.

The earlier portions of this case-history should be studied in contrast to that of Case IV, and the management of them noted in the light of their subsequent course.

The painless type of mastoiditis is a form of middle ear infection to which reference has been made under Case II. It requires only a knowledge of its salient features to be easily recognized. *A profuse discharge from the ear, disproportionate in amount to what could reasonably be expected to come from so small a cavity as the tympanum, generally running from the external auditory canal onto the cheek, and at night, or when the patient is lying down, making a pool of pus on the pillow, with little or no mastoid tenderness, is all that is generally present, subjectively or objectively.* The factors producing this type, its more characteristic bacterial flora and its other etiologic elements are not germane to the purposes of this report and are omitted here. What is desired here is to point the lesson that for nearly three weeks this patient evidenced the characteristics of this type of lesion. Consequently, the therapy indicated, viz., surgery, was not invoked until too late to save life. The contrast between the outcome in Case II and that in Case V is, in the writer's opinion, mainly due to the recognition of the gravity of this type of infection in Case II and failure to grasp its significance in Case V.

Later in the course of the history, the absence of pain was stressed to the writer when he urged operation, and consent was not readily conceded until the positive blood culture made reasonably certain that a grave complication was already present.

When the clinical picture changed, the absence of discharge was not understood in its relationship to the development of the lesion in the mastoid and blood vessel. In the presence of very high fevers, it is the writer's observation that ear discharge very often lessens in quantity or is entirely absent. Conceivably the mastoid lesion had run its course in a measure and the temperature then had its origin in the blood vessel lesion—the thrombosis.

Therapy was limited to opening and reopening the drumheads, because it was reasoned that only the ears gave any symptoms at all to account for the temperature. A mastoiditis was not believed to be present, and certainly no idea was entertained as to there being a sinus thrombosis present.

One should not speculate *post hoc ipso*. Yet it is justifiable to ask what the probabilities might have been had the painless type of mastoiditis been recognized and the fact been given due weight that primary jugular bulb thrombosis sometimes occurs in cases which show little mastoidal invasions and often give hardly any symptoms from the mastoid, and had there been, instead of repeated drumhead incisions, repeated blood culture studies and blood cell examinations during the three and a half weeks of septic temperatures and increasing prostration.

The case is presented to point the lessons regarding sinus thrombosis, and no discussion of the questions involved in the meningeal aspects of the case will be held here except to say that the case, in the opinion of Dr. Emil Altman, Dr. Julius J. Hertz and others who saw it at the hospital after operation, presented clinically the picture of an encephalitis lethargica; but this diagnosis, in the face of the finding of the pneumococcus in the cerebrospinal fluid, was untenable.

Finally, it is to be noted that the pneumococcus was found by the writer in the discharge from the ear when he first saw the case, and in the cerebrospinal fluid later when meningitis was developing. From the blood culture, the streptococcus hemolyticus, only, was obtained.

Case VI. Lateral Sinus Thrombosis Involving the Jugular Bulb in a Case of Bilateral Mastoiditis, Painless Type—Determination of Side Involved—Secondary Operation on Bulb—Temporary Facial Paralysis—Postoperative Otitic Sepsis with Abscess Formation in Muscles—Pulmonary and Abdominal Symptoms—Blood Transfusion—Recovery.—On March 14, 1920, R. K., female, aged 6 years, came under the writer's observation, giving the following history:

Early in February the child complained of a feeling of fullness in the head which was soon followed by a profuse purulent nasal discharge. There was no elevation of temperature, and local and general treatment were administered and the patient kept quietly in bed.

On February 15 there was a rise in temperature to 101°, and the next day the right ear began to pain. This increased in intensity, and, with a rise in temperature to 104° the following day, necessitated an incision of the membrana tympani by Dr. Murray Bass, who was called into consultation to see the case. Relief ensued, and a discharge from the ear was established. The temperature dropped to 102°, and the patient was relatively comfortable.

On February 19 the left ear gave symptoms, and the temperature was again 104°. Dr. Alfred Braun opened this ear, and for the ensuing three weeks the child was treated and kept in bed, the ear douched, and watched by the local physician. The patient seemed comfortable, but the discharge from both ears was very profuse. There was no complaint of pain, and the temperature range was low, between 100° and 102°. The nasal discharge had meanwhile abated.

When he again saw the case, on March 13, Dr. Bass found both membrana tympani markedly bulging and an absence of tenderness or other classical symptoms of mastoiditis; yet the following day showed an abrupt rise of temperature to 105.8. The writer saw the patient at this time.

She presented the typical picture of the painless type of acute mastoiditis. Both ears were discharging profusely. There was no tenderness on pressure over either mastoid process, yet the discharge was coming away under pressure-pulsations, and there was a noticeable drooping of both posterior external canal walls.

The diagnosis of double acute mastoiditis, painless type, was made, and immediate double simple mastoidectomy was urged. A culture was taken from the discharge, which was subsequently found to contain pyogenic streptococci.

The child was taken to the Stern's Private Hospital; and, on admission, the general physical examination was negative except for the condition in the ears. The temperature was 102.4°, respirations 30, pulse 118. Dr. Frederick Whiting was asked to see the patient and concurred in the opinion as to the condition and the advisability of immediate operation.

Double simple mastoidectomies were therefore performed.

Right Side. Coalescent type of lesion found. The intercellular bony structure was found to be mostly destroyed. Pus and detritus

filled the mastoid process. No sinus nor dura was uncovered at operation.

Left Side. A condition similar to that upon the right side was found. In addition, the inner table over the lower portion of the sigmoid sinus was found necrotic, and this was removed. As far as it was determinable, the sinus wall seemed unaffected.

The skin wounds were partly closed by sutures and the usual dressings applied.

The reaction from the operation was very prompt. A few hours after operation the temperature rose abruptly to 104°, and remained between 103° and 104° most of the following day. Pulse was variable, between 128 and 140, and its quality was not very good. At times it was weak and thready. About midnight of the 17th, the temperature dropped to 99°, as abruptly as it had risen, but the pulse rate remained high, 140. Respirations 26.

The dressings were soaked, drainage from the middle ear profuse, and the general status of the patient was extremely irritable. Sleep was absent, the patient dozing only at long intervals. General physical examinations at this time were negative.

The next day showed no changes of moment, except that there was complaint of persistent headache; and because of the profuse saturation, the external layers of the mastoid dressings were changed. During this procedure, it was noted that the wounds gave an unusually profuse amount of secretions, some of which came away over the skin of the surrounding area. The temperature at this time ranged between 99° and 102°, highest in the afternoon; the pulse, of better quality, was between 110 and 120; respirations 26. March 18 gave a similar picture, but a slightly lower temperature. Cephalalgia was still the chief complaint, and the unusually profuse wound discharge was the most salient feature presented by the examination that day. It was impossible to definitely localize the cephalalgia.

On the morning of March 19 the temperature was low, pulse 118, and respirations 24. The dressings over both mastoid wounds were completely saturated, and pus was exuding from them over the cheek and neck. A complete change of dressings was made. The left mastoid wound was found relatively clear, although there was more wound pus than usual present. On the right side, however, the wound seemed literally bathed in pus. The superficial tissues overlying the excavated bone showed sloughs along its edges, and lymph nodes, thickened and, in places, broken down. These were cleared away after all sutures had been removed, and wet dressings applied.

For the next few days the local conditions improved, although a slight rise in temperature was noted. Cephalalgia was still the chief complaint of the patient, who seemed unable to sleep during the night, and whose irritability was marked during the day.

Meanwhile the local treatment of the wounds and the daily changes of the mastoid dressings had controlled the infection in the mastoid wounds. The wound secretions were now about what is usually to be found, but the cephalalgia and irritability of the patient continued, and the temperature gradually mounted. On March 22 it reached 102°, dropped to 99° and again rose to 102°; and on the 23rd it reached 103.6° to 104.2°. The pulse was weak and thready, averaging 128; the respirations were 24. The general appearance of the child was not good, periods of restlessness alternating with those of drowsiness. Dr. Murray Bass made a thorough physical examination which gave negative results.

A blood examination made by Dr. Lester Unger gave a total of 15,500 leucocytes, of which 80 per cent were polynuclears, 15 per cent small, and 5 per cent large, mononuclears. A blood culture was taken, to be subsequently reported as positive, the bacteremia being due to streptococcus hemolyticus.

The temperature eventually reached 105°, pulse 132 and respirations 24 to 30.

It was recognized that the patient presented a number of serious problems, all of which had to be correctly solved if life was to be saved.

The negative general physical examination, with the temperature curve and the finding of streptococcus hemolyticus in the blood, pointed to the diagnosis of septic sinus thrombosis. But the determination of the side involved, and the question as to the ability of the child to withstand the shock of surgical procedure in view of the weak cardiac condition as evidenced by the pulse, left the writer in doubt as to the advisability of undertaking the surgery indicated by the clinical picture even were it possible to correctly localize the side which presented the thrombus.

A conference was called, attended by Drs. Emanuel Libman, Wendell C. Phillips, Murray Bass, Charles H. May and the writer.

Another general examination gave negative results, and the diagnosis of septic sinus thrombosis was concurred in. Dr. May, whose expert opinion on the eyegrounds was asked, stated his inability to find any differences existing in either the right or left optic discs which would warrant deductions to determine upon which side to intervene.

The findings in the mastoid processes at the original operation were all the facts left upon which to base a decision. In addition to this, it was decided that, in the event of there being no other findings which would be a guide, equal measured amounts of blood would be drawn by puncture from both sinuses and carefully plated, and the side giving the larger number of colonies was to be considered the one upon which resection of the jugular and exploration of the lateral sinus was to be undertaken.

A careful inspection of the wounds was made by both the writer and Dr. Phillips. The right mastoid wound was still the one showing the greater amount of pus, and there still remained signs of local infection of the soft parts here. The left wound was cleaner, the wound discharge more moderate in amount, and such few granulations as had made their appearance were apparently more healthy looking than on the right side.

In contrast to these present findings, we had the notes of the findings on both sides at operation to give additional data for consideration. These showed that, at the time of the original operation, the sinus wall on the left side had been found to be necrotic and was removed, exposing the blood vessel for some distance below the knee toward the jugular bulb. Because of this, and in spite of the local evidence of infection on the right side at this time, it was determined to first explore the left lateral sinus. In this decision Dr. Phillips concurred.

Operative Findings. After removing the granulation tissue and bringing the exposed lateral sinus wall into view, it was marked that pulsations of the sinus were absent. When the needle of the aspirating syringe was inserted into the sinus, it was found that no blood could be drawn into the syringe. The exploration of the sinus was then determined on at once.

The usual procedures were undertaken and a thrombus found extending into the jugular bulb. No bleeding was obtainable at all from the bulbar end. Extending the sinus wall backward only a short distance toward the "knee" brought a free hemorrhage.

The patient's condition seemed very poor, the pulse was very thready and irregular; and, upon the advice of Drs. Libman and Bass, further operative procedures were discontinued and no attempt made to separate and ligate the internal jugular at this time. The medical consultants felt that to continue further surgery at this time would imperil the child, and defeat the end sought.

It was decided that, in view of the necessary secondary operation, a blood transfusion should be done first so as to sustain the patient

and add blood factors to hold the systemic infection in abeyance.

The temperature abruptly dropped to 99.8°, the pulse 120, and respirations 28. The temperature, however, was on the rise again the next morning. Dr. Lester Unger performed a direct transfusion from a young vigorous soldier to the patient, the bloods of donor and patient having been matched meanwhile. 250 c.c. of blood were given. Late afternoon found the temperature at 105°, pulse 132 and respirations 28. Toward evening it had receded somewhat, and, fearing another high elevation, it was determined to proceed at once to the unfinished jugular surgery.

A speedy ligation of the jugular was performed, time of operation being a factor now. Two ligatures, a short distance apart, were thrown about the vein and the incision closed

Second Postoperative Period. For some time after the operation the pulse was thready, but its quality steadily improved. The excitability and irritability of the patient were gone, and in their place drowsiness was exhibited.

On March 26, the day following operation, the temperature ranged high, between 104° and 105.4°. The pulse was between 130 and 148, and there seemed to be a marked increase in the respiratory rate, which was about 44. The cardiac condition continued to give concern. The pulse was thready, occasionally intermittent, and weak. The respirations were shallow and rapid, but there was no further complaint of cephalalgia, nor other discomfort.

It would prolong this long case-history to record each and every medical measure instituted by Dr. Bass to sustain and support the patient during this critical period, nor is the writer even attempting to outline the medical features of this case. What is desired is simply to present a running account of the salient clinical features of the case, and the problems, both medical and surgical, which this patient presented during her long illness, so as to give data for an intelligent comprehension of what was done and the reasons which prompted the treatment.

During the ensuing night, sounds suspicious of pulmonary edema were heard over the chest, and it was thought that the fight was doomed to failure. However, under administration of caffein, whiskey and atropin, this was finally controlled.

A blood examination was made and gave 19,500 leucocytes, with a differential of 89 per cent polynuclears, 9 per cent small, and 2 per cent large, lymphocytes. The red cells numbered 3,500,000, with a hemoglobin index of 70 per cent. The index had dropped to 40 per cent before the transfusion.

On March 27 the temperature ranged between 102.4° and 103.8°. The child slept at short intervals. When awake she was irritable and very restless, and complained of pain in both ears and in her abdomen also. Early in the day her condition seemed improved. The pulse was of better quality and less intermittent, but in the afternoon conditions reverted to the status quo.

A blood culture was again reported as positive for streptococcus hemolyticus; a differential blood count gave 24,800 leucocytes, of which 90 per cent were polynuclears, 9 per cent small, and 1 per cent. large, lymphocytes.

The following morning conditions were about the same; temperature range 103° to 105.6°, pulse 130, respirations 44. The breathing was short and labored, and the pulse thready. The child was occasionally irrational. The wounds were dressed and the bulbar end of the sinus wound showed that the thrombus was beginning to break down; and fluid pus was obtained from the bulbar end of the opening into the sinus when pressure was exerted over the neck.

On the whole the patient apparently was worse, and Drs. Libman, Bass and the writer called Dr. Frederick Whiting into consultation.

In view of the second finding of a positive blood culture and the apparent breaking up of the thrombus in the dome of the jugular bulb, and in the absence of any secondary lesion's being found in the heart or lungs by Drs. Libman and Bass, it was Dr. Whiting's opinion that the dome of the jugular bulb should be laid free and all reachable thrombus particles removed. To this procedure the writer took exception, on the grounds that the general circulation was cut off from the main septic focus by the ligatures about the internal jugular, and that adequate drainage from the bulb was already in being, contending that even were the bulb of the jugular vein cleansed of thrombus particles, there still would inevitably remain the unreachable area beyond this point down to the site of the ligature. The discussion was academic, and because of Dr. Whiting's good results from the measure he advocated, the suggestion was adopted. The Grünert operation was not suggested, nor was it undertaken.

The patient was accordingly operated upon, the dome of the jugular bulb freed as far as possible, and all reachable particles of thrombus removed. During these procedures, the bony covering of the facial nerve coming into view was traumatized, and signs of a left facial palsy were found immediately after operation, only the upper branch being uninvolved and showing normal activity.

Just prior to operation the blood showed 25,400 leucocytes, of which 91 per cent were polynuclears, 8 per cent small, and 1 per cent large, lymphocytes.

The child reacted well. The next morning the temperature was 101°, ranging about 103° during the day. The respirations were still labored but the patient seemed brighter. During the afternoon the patient exhibited a slight chill and the temperature rose to 104°, followed by a profuse perspiration. Cold packs and sustaining treatment were administered.

On March 30 temperature ranged between 100.8° and 101.4°. The pulse was good; the respirations, less labored, numbered 40 to 44. The dressings of the wound showed the right mastoid wound relatively clean, with only a moderate amount of discharge. The plugs in the sinus of the left mastoid were removed. The bulb was found clean, and only a small amount of pus mixed with blood appeared when pressure was exerted on the neck. The wounds were all dressed with dichloramine T in chlorozone.

All through the day the child was very irritable. Nourishment which up till now had been taken was refused. The patient talked constantly, often irrationally, and continually picked at her bed clothes.

Blood counts gave a total of 28,000 leucocytes, of which 83 per cent were polynuclears, 15 per cent small, and 2 per cent large, lymphocytes.

Stimulation and general sustaining measures were continued with success.

On the next day the temperature was between 102° and 104.2°, reaching its maximum in the early afternoon. Complete change of dressing showed the right mastoid wound to be granulating normally, but the granulations were paler than usual. On the left side granulations began to show in the bulbar region. Both middle ears were now dry. The neck wound was clean.

During the day severe pain in the calf of the left leg was complained of, and a wet dressing was applied. There was no swelling and no tenderness, but the limb could not be moved freely without discomfort to the patient.

On April 1 the patient was more comfortable and had slept quietly. The temperature ranged between 100.4° and 103.4°, rising gradually through the day and reaching its highest point in the evening. A blood culture taken on this day was subsequently reported *negative*. This was the first negative report to be received, and it encouraged everyone.

On the following day the temperature varied between 101.4° and 102.6°. The pulse was of good quality, between 120 and 128, and the respirations were between 30 and 34. In the afternoon the child became nauseated and the digitalis, which had been administered for some time, was discontinued. The nausea soon quieted.

The wounds were redressed and showed both mastoids to be granulating. The right middle ear was dry. On the left side the bulb was clean, but the middle ear was again slightly moist.

Meanwhile the pain in the left leg had continued and the tissues of the calf had swollen and become tender. Fluctuation was apparent, and an incision of one and one-half inches long was made into the abscess, about four drams of frank pus being evacuated from within the gastrocnemius. It was odorless. The incision was packed with dichloramine T.

On April 3 the temperature stayed between 101.4° and 102.6°. The pulse was from 120 to 128 and the respirations averaged 32. Stimulation of whiskey and strychnin was continued, with codein when necessary for control of the restlessness. The patient seemed more comfortable.

A blood count showed a total of 17,600 white blood cells, 92 per cent polys, 7 per cent small mononuclears and 1 per cent large mononuclears. There were 3,200,000 red cells and there was a hemoglobin index of 60 per cent. The color index was one minus and no abnormal red cells were present.

Very early the next morning the temperature was 103°. It dropped to 99° and then rose gradually through the day to reach 100.8° at night. The pulse varied between 118 and 128, and the respirations between 28 and 32. The child was fretful part of the day, but complained of no pain and was otherwise comfortable.

On the morning of April 5 the child's temperature was 99.6° in the morning. Throughout the day it rose and reached 102.8°. The pulse ranged between 120 and 134 and the respirations were 30. On this day the patient began to complain of severe pain in the region of the left shoulder, which was found tender to the touch, but there was no redness nor swelling, and a wet dressing was applied. The child was then comfortable and rested well.

Early the next morning the temperature ranged between 100° and 100.8°. Pulse was 118 and respirations 30. A small area of redness and swelling appeared over the acromial process of the left shoulder and this was incised under local anesthesia. An abscess was found deep in the deltoid muscle and about one-half of a dram of pus was evacuated.

That evening the temperature rose to 102.8°, the pulse ranged between 128 to 136, and the respirations were again increased and labored—between 32 and 34. Accompanying this, the child began to complain of an intense pain in the right side of the chest. She seemed to get relief only when she sat up in bed. A short dry cough was present. Codein and other medication were given to control the cough and relieve the pain.

Examination of the lungs by Dr. Bass showed that moist rales were present over both lungs, suggestive of a developing pleurisy and a bronchopneumonia, but the diagnosis was not definitely established because of the indeterminate character of the physical signs. All who saw the patient at this time believed that some lung condition was developing.

April 7, 1920, found the condition about the same. The temperature was 100.2° to 102.8°, pulse 120, respirations 34. The pain in the chest was still present and the physical signs were the same as the day before.

The character of the respirations was shallow and labored. Nourishment was refused and the child showed prostration.

The redressing of the ear wounds showed them clean and progressing favorably. The wound in the leg was now clean and healing, and the wound in the shoulder area also was progressing favorably.

April 8 showed the condition the same. Temperature 100.2° to 103.2°, pulse 128 to 136, respirations 30 to 34. Cough was still present.

During the next few days conditions remained the same. No definite diagnosis was possible. The dry cough was persistent; rales were evident over the chest, more in evidence on the right side. The temperature ranges were between 100° and 103°. The pulse was rapid and irregular, and the breathing labored because of the pain present when breathing. Some headache was also complained of.

On April 13 there was a severe paroxysm of coughing—temperature 101° to 104.8°, pulse 138 to 150. Both Drs. Libman and Bass were unable definitely to make a diagnosis because of the indefinite physical signs, and an expectant attitude and symptomatic treatment were continued.

A blood examination gave the following: The total leucocytosis was 18,000; polynuclears, 79 per cent; small lymphocytes, 18 per cent; large lymphocytes, 3 per cent. The total red blood count was 2,420,000; hemoglobin index 48 per cent. Slight poikilocytosis and anisocytosis were present.

The edema of the left side of the face and neck, which had appeared and then disappeared, was again evident, but was less marked on this day, and the facial palsy, which had become localized to the lower branches of the facial, seemed to be clearing, some motility of the mouth becoming noticeable. The child, however, seemed desperately ill and appeared generally worse than at any previous time.

The next morning the temperature dropped to 100.6°, but rose immediately to 103.6° and remained at this level throughout the day. The pulse ranged between 120 and 140, and the respirations averaged 36. A blood culture taken on this day was found sterile. The child's condition seemed precarious and a transfusion was performed, 250 c.c. of blood being given.

On April 15, in the early part of the day, the temperature was 101°, pulse 124 and respirations 32. The patient perspired freely and was comfortable, although she was nauseated for a short time and vomited once, a small amount of mucus. But in the evening the temperature rose to 104.2°, pulse to 148 and respirations to 48. The temperature reached 105.6°. The child was very restless and complained of severe headache.

Early the next morning the temperature was 102.4°. It dropped at noon to 97.4° and rose in the evening to 101.2°. The child vomited a small amount of mucus once, but was otherwise very comfortable. At 9 p. m. the temperature rose to 105°, pulse to 140 and respirations to 40, and the child was very restless.

On the morning of April 17th the temperature was 104°, pulse 136 and respirations 36. The child was fretful and restless. The strychnin was discontinued and caffein sodium benzoate gr. ii substituted as stimulation. That evening the temperature dropped to 101.4°, but during the night averaged 103.6°. The child complained of severe pain in the right leg and groin which was relieved by an ice bag. There were no external signs to account for the pain.

Early the next day the temperature was 104.6°, pulse 150 and respirations 36. The child was very restless but the temperature droppd to 102.6°, pulse 136 and respirations 34; and she slept well. A blotchy rash appeared on the legs which disappeared quickly when exposed to the air. In the evening the temperature dropped to 101.6° and then rose gradually to 103.4°. At midnight it reached 106°, pulse 160 and respirations 44. The patient complained of a severe pain in the right leg and groin. The thigh was held tightly flexed on the abdomen and any attempt at movement caused excruciating pain. Nothing definite could be made out by

palpation and there were no localizing signs of any sort, although the symptoms were those of a periarticular abscess of the right hip. Ice bags applied to the hip gave some relief.

The temperature on the morning of April 19 was 103°, pulse 136 and respirations 32. The abdomen was distended. In the afternoon the temperature dropped gradually to 99.8°. The signs in the chest were apparently localizing to the right side. There was marked dullness and distant voice and breathing. A radiogram of the chest was taken. It furnished no data of moment. The plate seemed to show the diaphragm raised on the right side.

The chest was aspirated, but no fluid found, and the diagnosis was still in abeyance.

That evening the temperature rose to 105°, pulse 150, respirations 44. At midnight the temperature had dropped to 103.4°, pulse 140, respirations 36. Cold packs were given and were well borne.

By next morning the temperature was 103°, pulse 134, respirations 34, remitting in the afternoon to 100.8°. The child complained of a severe headache, which was relieved by an ice cap.

That night the temperature reached 102°, and, on April 21, it reached 103.8°, with the pulse at 138 and respirations 36. The child still complained of severe pain in the right leg and groin. The leg was flexed on the abdomen.

Dr. Libman again saw the patient, and he now thought that the patient presented an infarct in the perirenal tissue with resulting psoas spasm giving symptoms referable to the hips.

That day a blood count showed a total count of 30,000 white cells, of which 85 per cent were polys, 14 per cent small monoes and 1 per cent large monoes. The red blood cells were 3,600,000—hemoglobin index 70 per cent. Color index was one minus. Aspirin gr. II was given every four hours and the caffein and whisky were continued.

On April 22 the temperature was 102.4° in the morning, dropped to 99.8° in the afternoon and rose to 102.2° at night. The child complained of severe pain in the right side of the chest, but was otherwise more comfortable. The aspirin was continued.

Conditions seemed at a standstill. The general appearance of the child was good. There seemed interference with movement of the right thigh. No point of tenderness was noticeable anywhere about the hip-joint; yet the patient held the right thigh flexed upon the abdomen and seemed most comfortable when in this position. The question of a septic focus in the area of the right hip-joint or the psoas muscle, or an abscess in the region between the liver and diaphragm, was discussed. Not enough data

was at hand to fix a diagnosis. Upon the advice of Dr. Joseph Blake, who was called into conference and who examined the patient, the expectant attitude was continued, and the case treated by meeting the symptoms as they presented themselves. He expressed it as his opinion that, with the findings at hand, no further surgery was indicated.

The child gradually lost all the symptoms complained of, the wounds all healed, the hip-joint lost its rigidity, and the chest signs disappeared. On May 25 the child was discharged from the hospital.

During the last few weeks the child was sent to Dr. Joseph F. Harris for electrical stimulation of the facial nerve and for stimulation of the muscles of the left side of the face. Under his care the last signs of the facial palsy disappeared.

The child was examined by the writer in July, and was found fully recovered.

The temperature chart is appended herewith.

COMMENT.

Like Cases II and V, this patient presented from the onset of her ear attack the painless type of acute mastoiditis. For a period of almost a month's duration there had been a very profuse discharge from the ears, inordinate if considered as emanating from the tympanic cavity only, accompanied by temperature. The case was identified as such as soon as seen by the writer, and immediate operation recommended. No time thereafter was lost, and the child was operated upon promptly that same evening.

Without going further into the discussion as to the reason why no diagnosis was made previously, suffice it to stress the point that both medical attendants and otologists must learn to recognize types of mastoiditis and comprehend the dangers which the painless type of mastoiditis presents. Reference is again directed to the comment upon this point under Cases II and V.

No one can say whether or not the sinus was already the seat of a mural thrombus at the time of the first operation. At least the operative findings at this operation disclosed a necrosed bony sinus plate on the left side. The vessel wall when examined at that time showed no abnormality of color or consistency, and no justification for exploring it existed. Promptly upon the advent of septic symptoms, the necessary steps were taken to ascertain the presence or absence of a bacteremia, and as promptly as the report was received, the operation on the lateral sinus and the jugular vein was undertaken. Yet, prompt as this was, it still gave sufficient time for the picture of an otitic sepsis to develop, with septic foci

in the muscles of the leg and shoulder, and all the rest of the clinical picture whose merest outlines are given above.

In the determination of the side upon which the thrombosis existed, every kown method to differentiate the side affected from the other side proved unavailing. The writer finally, relying upon the operative findings at the primary surgical procedures, explored the left side *first,* in spite of the fact that the right mastoid wound was infected and profusely discharging pus. Consultants who examine cases operated on by other otologists are apt to place too much reliance upon what they themselves find at the examinations they make. In the absence of any other governing data to cover the questions at issue, more reliance should be placed upon the surgical findings of the surgeon who exposed the interior of the mastoid process at the primary operation. Where such findings are substantiated subsequently by either the examination of the eyegrounds, the blood bruit in the respective jugulars or the comparison of measured blood cultures from both internal jugulars, then differentiation of the side involved is easier than in the above case, where all such data failed.

The secondary operation upon the dome of the jugular bulb which was done in this case presents a mooted question, involving a discussion of surgical judgment and an analytical study of the anatomy of the parts involved, together with a discourse on the theory of surgical drainage, which would hardly be in place in a paper designed to accentuate the clinical aspects of these cases. That presents a question purely otological and technical, the discussion of which is purposely avoided here. Whether much or little is to be gained by the small extra exposure of the interior of the jugular bulb obtained by this procedure, and whether or not a septic focus, which is already open externally and is draining, and which is already securely separated from the general systemic circulation by the ligatures placed about the internal jugular vein can be more securely isolated by this additional opening into the bulb, are questions which, at present, remain unanswered for the writer. Only a more extended experience will prove or disprove the wisdom of this procedure. Compared, however, with other cases of the writer, nothing was demonstrated in the subsequent course of this case to prove that any particular advantage accrued to the patient. The sepsis and the bacteremia continued unabated after the operation in question. In the other cases of this series where the dome of the jugular bulb was found involved, no secondary exposure of the dome of the jugular bulb was undertaken and the results speak for themselves.

The subsequent course of the case was medical. It presented the varying pictures of otitic sepsis. The painstaking care and attention to details in meeting the varying clinical symptoms were the problems which the medical attendants had to meet, and to them much of the credit for the saving of this life properly belongs.

In conclusion, the value of direct blood transfusion needs comment. In a hemolytic infection of the blood stream, where repeated blood cultures show that these streptococci are not only present in the blood, but are at consecutive examinations found to be gradually increasing in numbers of colonies, and the hemoglobin index is shown to be falling, no better sustaining agent exists, in the writer's opinion, than the injection into the patient's veins of whole, live, unadulterated blood from a donor whose blood matches that of the patient. In this case, after each transfusion, good was observed to result, and no doubt the repeated transfusions were a decided factor toward the eventual recovery from the septicemia, all active foci having been eliminated surgically.

CONCLUDING REMARKS.

I. From these and other cases, it is concluded that the painless type of mastoiditis is a source of potential danger to patients because its gravity is not recognized, nor is it often diagnosed.

II. Otitides of more than two weeks' duration which present a picture of sepsis otherwise unaccounted for should be studied with the possibilities of sinus thrombosis in view, and blood cultures should be taken.

III. In the event of repeated negative cultures from the blood, the clinical signs should govern; and exploration of the mastoid process and, eventually, exploration of the sinus should be undertaken before an agonal type of otitic sepsis, or a meningitis, develops.

IV. With primary jugular bulb thrombosis, it should be more generally understood that the mastoiditis which accompanies it is often negligible as to marked classical symptoms, and also as to demonstrable extent of lesion.

V. A more general knowledge of the various types of mastoiditis is desirable, as not all types are to be treated alike. Some present graver risks to the patient than others.

VI. Finally, the findings at operation should be carefully studied, as a hemorrhagic lesion, accompanying or following an attack of influenza, and giving septic symptoms, should make a survey of the sinus condition imperative as soon as the necessary data to establish the diagnosis are at hand.

51 West 73rd Street.

THE LOGICAL CAUSE, PATHOLOGY AND TREATMENT OF BRAIN LESIONS—AN EXPERIMENTAL PAPER.*

Dr. Alfred Kahn, New York.

At different times over a period of a number of years, the thought has repeatedly come to my mind in thinking of brain lesions from otological origin—why it is that many of these cases die either where they have had operative treatment or where nothing of a surgical procedure has taken place, and why it is that many of these cases recover, even where no therapeutic defense is brought into play. This latter class of cases of brain involvement just simply recover. Cases of brain abscesses have repeatedly been diagnosed as brain abscess only on the operating table. Their presence was in many cases not even suspected. Brain abscesses have been found on the post mortem table, where the patients have come to their end through some other intercurrent condition. Brain abscess cavities have been found to be filled with a purulent substance walled off—this purulent substance was found to be bacteriologically sterile. Can it be doubted that at some previous time this purulent appearing substance was infectious and of pyogenic origin?

It has often seemed to me—and I have repeated it here at various times, in discussion, that our surgical principles as practiced were not surgically logical; that our procedures, although the best we had at hand, were inadequate, that we often, for example, opened the sub-dural space and did a decompression operation, that we often explored for brain abscesses where no brain abscesses were found, that we did these things surely because we thought them advisable, and largely because they were the only means that we knew for combating these complications. Do not misunderstand me—I know that we have arrived at these procedures through years of development, clinically, surgically and experimentally, and I have great respect for these procedures. It is only with an intense desire to give something, to do something that will perhaps precede or go hand in hand with this knowledge which we have so laboriously accumulated, that I have interested myself in the matter.

This subject is an extremely difficult and broad one, and in this comparatively short paper I am trying to group a variety of differ-

*Read at a meeting of the Otological Section of the New York Academy of Medicine, May 14, 1920.

ent complications—I mean brain complications of otological origin. This, in substance, chiefly refers to infectious meningitis and brain abscesses.

Among the first questions which arose in this experimental work was what is meningitis—what is brain abscess—how do they occur—what is the pathology of these lesions? Briefly stated, I concluded that meningitis, brain abscess—were the result of infections by a pyogenic germ or germs—and these experiments will endeavor logically to show that—first, let us say for example, in meningitis, the germ attack is made at some particular spot of the meninges, that the point of attack is more intense (that the irritation is more intense—that the reaction is more intense) at some one point on the surface of the meninges than at any other point. Logically, there must be a spot of beginning. This is the point of invasion, this is where the battle starts. The system, either immediately or after a period of time (an incalculable space of time, an infinite space of time) reacts according to its ability, and you have a state of inflammation, a state of congestion, exudation, and transudation and absorption. And now your clinical symptoms start (fever), a change in temperature, either first a rise or fall. This may not be noted in the clinical chart; this is followed almost invariably by a rise in temperature and you have your clinical vibrations—the character of the blood begins to change—change in number of leucocytes, quantity of blood, consistency of blood, presence of antitoxin and immune bodies—at first gradually, then in increasing amounts, finally the presence of toxins, then germs in the blood—at first dead, finally as death nears, living germs.

These clinical symptoms and signs and reactions on the part of the system you all are acquainted with, but what I want to especially point out is that you have two processes taking place in an infection of this character. That you have first a local process, an inflammation at the point of invasion, that the system at this point of infection is continually trying to localize, to surround the point of irritation. When a patient recovers, what in reality takes place—the point of infection is localized and absorbed, or it may be only localized without absorption, and you have your brain abscess for example. Now this brain abscess may remain infectious, quiescent, or the sting may be taken out of the infectious substance by the immune activities of the system, and you have your sterile pus cavity. I do not believe this latter class to be dangerous. In addition to this localized point, the debris absorbed from the point of battle is taken up into the system and you have your

toxemia, and the system forms antitoxins and throws out immune bodies, and inaugurates other means of defense. Now, if the defensive measures inaugurated by the system are strong enough to overcome the activity of the invading organisms, your patient recovers. If the activities of the invading germs are overwhelming, your patient dies. Now what I want to particularly point out and especially call your attention to, is that the patient does not die from the local process, unless of course, the localized area should cause pressure on vital centers—but that the patient dies from the absorption of poisonous toxins from the localized area of battle. This you might dispute, but I will show you through a series of experiments performed within the peritoneal cavity on the peritoneum, where there are no vital centers to be pressed upon, that the same thing takes place and that the patient does not die from the local spot, but that the main thing is the poisoning of the system through absorption. Now you must see that this could not logically be otherwise, and you must therefore also see that our final great success in treating these complications must come through the general system, more so perhaps than at the point of local invasion.

I will not take up your time unnecessarily by enumerating each experiment separately, and going into the detail of each experiment. These experiments were carried on in the laboratory of Experimental Surgery, New York University, and the conclusions arrived at may have been the result of one experiment or they may have been the results of experiments on several dogs, or finally it is the impression made upon my mind, drawn from these conclusions at this writing. First, I learned that absorption from the sub-dural space is quicker than in the peritoneal cavity—that in operations upon the brain the element of shock is very much greater than in operation in the peritoneal cavity, that reaction upon the part of the system to infections within the brain are slower in comparison to infections in the peritoneum, but absorption is quicker. My primary object in doing these experiments was to learn first if I could infect a dog and allow him to go to a point where death from infection seemed imminent, and then to see if I could not bring into play some force that would keep the dog from dying. I first opened the peritoneal cavity of the dog, and took dust from the laboratory room and placed it in the dog's peritoneal cavity. Where dust was used in this way the dog practically invariably recovered without any interference on my part. The germs streptococcus and staphylococcus from the laboratory (I call your attention especially to the fact that I mention laboratory germs) were injected into the peri-

toneal cavity and into the blood stream of dogs and nothing happened. I took small pledgets of gauze saturated with pus taken from dogs that had just previously died from a purulent infection. I then took pure pus without being saturated with gauze (liquid pus) and put it in the peritoneal cavity. This liquid pus was taken from dogs that had just previously died from a purulent infection. Where the pus alone was used, the dogs became immediately very sick and many of them died within twenty-four hours. Post mortem: In many instances showed no reaction whatever. In other instances where pure pus was used, there was evidently an endeavor on the part of the system to react and throw up a defense, for the peritoneal cavity was intensely inflamed and purulent. When gauze saturated with dust from the room was used, the systemic reaction was very slow, the dog would recover, and on post mortem, would show the gauze walled off. Where gauze saturated with pus was used, the reaction was more intense and quicker than where gauze saturated with dust was used. These dogs became quite sick, but some of them recovered and on post mortem in this class the gauze would be either entirely walled off or the walling off would be very far advanced; that is, if the dog had recovered, and the post mortem done some days later, the gauze would be entirely walled off, but in those dogs that only lived a few days, after the original infection, the gauze would be in various stages of being walled. Where pure pus was used, the peritoneal reaction was not comparatively as great nor did the tissues to the naked eye seem to localize at all. Of course, the previous experiments had a foreign body, which acted as both an irritant and a nucleus around which to localize, but in those cases where pure pus was used, the systemic reaction took place almost immediately, and these were the hardest cases to handle, as very often the dogs would die from toxemia before any treatment could be started. The practical conclusions to be drawn from these experiments are: First, that it is not the infectious organisms by themselves alone or dirty material containing these germs, of a non-animal character, that is so dangerous, but what really causes the toxemia and final death in these cases of peritonitis, meningitis, brain abscess, etc., is the absorption of the debris resulting from the strife of the tissues on the one hand and the invasion of the infectious organisms on the other. Secondly, that clinically speaking, the more important process is the systemic toxemia. This is what causes the final death. It is not the local process, for the local process is finally, if at all, overcome by systemic activity. Thirdly, that any serum treatment to be found in the

future will owe its success or failure to overcoming the debris resulting from this infectio—inflammatory combat, and that it will not be based upon the activity of germ life in the laboratory. In other words, it will probably be an autogenous serum. Before proceeding further, I overlooked stating that we injected directly into the blood vessels of some of these animals weak solutions of citrate of soda and blood that were not sterilized—solutions that were surely filled with all forms of bacteria—and that the animals were not in the least affected.

By way of remark, it may not be out of place to draw further conclusions from these experiments. It must be remembered that when we introduce pure pus into the peritoneal cavity, or into the sub-dural space, pus from another being is not the same thing as pus being formed in a subject himself—it is a more toxic test. In the latter case, before pus is formed, the system has gone through the various stages of irritation, inflammation, localization, breaking down and absorption, absorption only taking place when the pus is formed. In the first case, however, the economy does not go through all of these various stages, but pus is introduced directly as pus, without having been manufactured at any point of localization. Therefore, the body has had no chance to react and no chance to form antitoxins. Absorption takes place directly and your patient is poisoned before antitoxin can be formed. And this absorption took place so quickly from the sub-dural space, that the few dogs upon which I tried it, died before I could do anything with them. The absorption from the peritoneal cavity is slower and the system has some chance to form antitoxin and attempt to immunize itself. These conclusions may not be absolutely correct, but it is the only way I have been able to interpret them.

Treatment: The next question that now arises is how do you propose to cure such a state of affairs. The procedures now used are practically all surgical and are applied to the local lesion, and they consist in cases of meningitis, primarily of decompression; this is done chiefly to give the brain tissues more room to expand; it doubtless has value in this regard. As to the value of an early or late decompression, I do not think that we are at all sure. To my mind, in one instance, a decompression may be of value; in another instance, it may be of little value, or of no value at all. I believe that our success in treating these cases in the future will develop along lines that have a tendency to immunize the system to the invading organisms until the cells in the localizing area are able to localize the infection, and I can only see two possible ways of

meeting success. Although it is not intended to cover the first in this paper, I think it should be mentioned. I believe that it may be possible to make a serum that will be effective. The germs entering into the formation of this serum will have to be propagated and fed on human blood or human tissue. In cases where meningial or brain tissue is involved, if the germs entering into the formation of a given serum could be fed on brain tissue or meningial tissue, or upon the blood of the patient himself, I think the serum offers a chance of success. I do not believe that germs cultivated on any other artificial media will have a chance to succeed. The second method is covered by this paper, and I believe offers our first immediate chance of success. And this method is a method of transfusing blood from one person to another and back again, over a period of time or at frequent intervals, and is a method which I described nearly ten years ago, and which is covered by these experiments, as follows: At the start I want to state so that there will be no misunderstanding, that this method has never been tried on human beings. The work following is purely experimental on animals, but the success of these experiments has been of such a nature that I believe it would be perfectly safe on human beings.

My experiments consisted of infecting the peritoneal cavity of dogs in the various ways that I have mentioned before. The dogs were infected with pus from other animals, which had previously died of the purulent infection in question. I soon learned that within twenty-four to forty-eight hours a dog would reach the height of his infection, and that if he were not transfused in the way in which I have described before this elapsed time, that he would die, that those dogs that were transfused within a period of twenty-four to forty-eight hours, that they made either a total recovery or that they lived several days longer than those dogs which were not so transfused. That when the peritoneal cavity was infected as described, the dogs transfused according to this method would almost invariably recover, but when they were not transfused, they would die within a period of forty-eight hours to several days. The technique of these experiments was as follows: On the first day the dog's peritoneal cavity would be opened and the infectious material inserted. After an interval of twenty-four to forty-eight hours, depending on the nature of the infectious substance, but chiefly on the condition of the dog, he would be transfused. The transfusion in several instances was continued for a period of from thirty minutes to two hours, a continuous circulation of blood between the two animals.

The next question is apt to be, what takes place when the circulation of blood is continued between the two animals? I have already shown you that during the state we clinically call meningitis, that there is a battle for existence and that there is an absorption of toxins, and there is a manufacturing of antitoxin—and the system is trying to immunize itself. Now, let us say, that the toxins have the best of it, that the system is loaded with toxins. Now if you can take away part of these toxins you are reducing the poisonous dose just so much. When you transfuse in the way I suggest, this poisonous blood passes over to another animal, and if the transfusion is continued, it reduces the poison in the infected animal just one-half, because it must be remembered that while this toxic blood is passing over to the healthy animal, that an equal quantity of healthy blood is passing over to the diseased animal, in each instance, having been reduced one-half, and the toxin kept below the point of a poisonous dose. At the same time, it must be remembered that the blood from the diseased animal to the healthy animal contains substances which will cause the healthy animal's organs to act and react and form an additional amount of immunizing substances, thereby increasing the force of resistance, let us say, perhaps another 50 per cent. The diseased animal is reinforced with an equal quantity of healthy blood, blood which is further capable of manufacturing immune substances, thereby increasing the diseased animal's chances still more. If the diseased animal, after a lapse of twenty-four to forty-eight hours, is again transfused with the same healthy animal, his blood is still further reinforced by antitoxins. This can be repeatedly kept up until the local point of infection is walled off and the diseased animal able to take care of himself. In other words, by frequently repeating the transfusion the diseased animal is put into an antitoxic state, following an exact example set by nature, and he is finally absolutely cured. Now, what takes place in the healthy animal? We are not injecting pus from the diseased animal to the healthy animal—you are injecting a substance called toxins, if you will, but of a dose insufficient to kill. You are not necessarily injecting infectious organisms, and even if you were injecting free organisms, the organisms without the debris are not so apt to affect the healthy animal as you may imagine. The debris—as I have pointed out to you—is the substance which kills, and that is represented by the toxemia. In this connection, I wish to state that I did not lose a single donor from infection. The healthy dog never developed an alarming toxemia, and none of them died from infection. Another question that may

arise is, why wouldn't saline solution or a direct transfusion of blood—why wouldn't these two methods be just as good as the continuous transfusion method that you advocate? I believe that both of these methods have some value in this connection, but I do not think it is very great, for this reason: If saline solution were injected into the blood stream in any quantity, it is true that it would dilute the toxins, but it is also true that it will reduce the antitoxins also, the relative quantity of toxins still remains, and the relative quantity is what kills. It is also a question as to whether saline solution by its stimulating effect, would not cause faster absorption. Now, as to the direct transfusion of blood, to a certain extent the same thing takes place as takes place with salt solution. Both the toxins and the antitoxins are diluted, but even if the antitoxins were not diluted, do you think you could introduce a sufficient amount of blood to really amount to anything? The relative quantity of toxins remains; and then you cannot dilute the vessels to the bursting point, or you are apt to have cerebral hemorrhage, or an engorged heart; so in the last analysis, you can readily appreciate that the method I describe is the only one up to this writing, which offers any chance at all. The diseased toxic blood is removed and replaced by healthy blood, quantity for quantity, by a method which can be continued for an almost indefinite period without loss of a drop of blood—one more than the other—if necessary, to either animal.

50 East 42nd Street.

LATERAL SINUS DISEASE—CASE REPORT.

Dr. H. H. Martin, Savannah, Ga.
Dr. S. J. Crowe, Baltimore, Md.

A boy of twenty years had had scarlet fever with otitis during the first year of his life, the latter condition continuing as a chronic process in each ear without complications until April, 1919, when he suddenly developed mastoiditis on the left side with a sub-periosteal perforation. This was the first time he had come under my observation. A simple mastoid operation was done by Dr. S. J. Crowe, about May 1, with a very satisfactory result. He returned to me about June 1 and by July 1 the wound was entirely closed and the ear on the operated side was perfectly dry and remained so up to the time of his death. The right ear, however, continued to discharge under the same treatment used in the left, namely: daily application of saturated solution of boric acid in alcohol. I did not see him again until September; at that time the condition was essentially as described above. He was instructed to continue the local application on the right side and return in four weeks. This he did not do, remaining away until January 14 this year, when he came in complaining of frontal headache and pain in both eyes, which had existed for one week; declared that his ears were all right, neither of them discharging. Examination revealed that the right ear was anything but all right, the canal being entirely filled with a large granuloma. The fundus in each eye was normal, pupillary reactions normal but right pupil was slightly larger than the left, temperature 98.4°, pulse 78, general condition apparently normal with no complaint excepting frontal headache and pain in eyes. He was ordered to the hospital for removal of the granuloma. On awakening the next morning he had a slight chill with nausea and vomiting. I saw him at 12 o'clock noon and he seemed perfectly all right, showing no further symptoms of serious trouble; the blood count, however, showed a leukocytosis of 23,000.

On the strength of this rather indefinite group of findings I decided to do a simple mastoid operation in addition to the removal of the granuloma. This was done during the afternoon of January 15. The mastoid bone was found completely eburnated, being flinty hard and devoid of cells; neither pus nor necrosis were found until I had cut down to the inner plate, when a necrotic groove was un-

covered leading down to the tip, which showed a necrotic area at that depth. There was no visible communication between the tympanic cavity and the mastoid bone, and no antrum could be demonstrated; therefore, an opening was made through the flinty hard bone directly into the tympanum and the granuloma was removed with gentle curettment of the tympanic cavity. The wound was closed with cigarette drainage and the patient to all appearances entered into a perfectly satisfactory convalescence, exhibiting the usual post-surgical temperature for three days followed by a period of six days, during which time the blood count dropped to 8,800 W. C.; temperature, pulse and respiration became normal, wound healed by first intention, stitches and drainage removed on the fifth day. Patient up and around in a wheel chair, and in every way giving indications of a perfectly normal convalescence.

However, during this time the patient complained of stiffness and pain in back of neck on two occasions, although this could not be demonstrated objectively. The first time was during the second day following the first operation, the second was during the eighth day and was coincident with a slight chill, a rise of temperature to 101°, with a rather slow pulse, and a W. C. blood count of 25,700, 85 per cent poly and 14 per cent lympho. Two days later the blood count was 20,400 W. C., 89 per cent poly and 11 per cent lympho, with a typical septic temperature which, however, did not rise above 102° nor fall below 98.3° until the eleventh day after operation, when there was another slight chill with nausea and a blood count showing W. C. 22,000, 92 per cent poly and 8 per cent lympho. There were no fundus changes visible at this time and no other definite clinical findings. However, I reopened the mastoid wound on this date curetted the surgical cavity and cut out the entire posterior canal wall, giving free access to the tympanic cavity without disturbing the ossicles or the remnant of the drum membrane. No pus was found at this operation, but there was revealed a small area of necrosis deep down on a level with the internal tympanic wall. This was cautiously curetted, but the sinus was not exposed. There was immediate improvement following this operation as after the first one. Blood count twenty-four hours after the operation, W. C. 17,000, 86 per cent poly and 15 per cent lympho. This improvement continued for about ten days, during which time he complained only occasionally of pain and stiffness in the neck, but the blood count remained high in white cells, varying from 17,000 to 20,000. The drains had been removed and the wound closed on the eighth day, but two days later pus appeared in

the mastoid wound for the first time, the lower end was reopened and a drain inserted. Patient was up in a wheel chair and was feeling well(but complained more and more of the pain and stiffness in the back of the neck, temperature after remaining at normal for about one week again showed daily elevations to 101° but was never subnormal. This condition continued for a period of ten days after the second operation and was clearly getting no better. At this time Dr. S. J. Crowe, who had operated on the opposite side, was called. Dr. Crowe has kindly consented to collaborate with me in this case report, and his contribution follows:

* * * * *

The patient did not look very sick. His only complaint was a constant dull pain in the occipital region on the right and some stiffness of the neck. He was in bed when I saw him, but for the past week had been spending a part of each day in a wheelchair.

The general physical examination was negative (Dr. Waring).

The examination of the nose, throat, teeth and accessory nasal sinuses was negative.

The neurological examination showed:

1. The right pupil was larger than the left. Otherwise the second, third, fourth and sixth nerves were normal.

2. There was no spontaneous nystagmus and no other symptoms of a cerebellar lesion were noted.

3. Loud conversational tones could be heard on the right and the vestibular reactions were normal on both sides. (Caloric test.)

4. The ninth, tenth, eleventh and twelfth nerves were normal.

5. The reflexes were equal and active. There were no motor or sensory disturbances aside from a weakness of the facial nerve on the right. This first appeared shortly after the last mastoid operation and was probably due to operative trauma.

6. Kernig's sign was negative. The neck could be flexed till the chin touched the sternum but this was quite uncomfortable.

7. The only general pressure symptom was the presence of a definite choked disc on both sides. The retinal veins were dilated and the nasal margins of the discs obscured to about an equal extent on both sides.

8. The veins in the eyelids, temples and forehead were dilated, but equally so on the two sides. There was no tenderness on pressure along the course of the internal jugular veins.

The most striking positive finding was as follows: Pressure on the left jugular vein causes a marked increase in the dilatation of the veins of the fundus (as seen with an electric ophthalmoscope),

and a marked increase in size of the superficial veins of the forehead, eyelids and temples. All these dilated veins immediately empty when the pressure on the left internal jugular vein is released. Similar pressure on the right internal jugular has no appreciative effect on either the retinal or the superficial veins.

If both internal veins are simultaneously compressed the superficial and retinal veins immediately fill up; release of pressure on the right has no effect; release of pressure on the left causes them to immediately empty.

This clearly demonstrated that there was an obstruction to the outflow of blood on the right side, and that this obstruction was located either in the lateral or sigmoid sinus or in the bulb of the internal jugular vein.

The operation disclosed a thrombosis involving the upper third of the sigmoid and all of the lateral sinus on the right. The sinus wall was everywhere covered with granulations. The internal jugular and facial veins were first ligated. There was no evidence of infection in this region. The whole of the sigmoid and about two-thirds of the lateral sinus was then freely opened. The lateral sinus was filled with pus and granulations. These were removed, but we were never able to get a free flow of blood from the cerebral end. This was discouraging, but on account of the boy's general condition, and the necessity of removing the towels and shaving a larger area before it was possible to expose the remainder of the lateral sinus, it was thought best to discontinue the operation at tion this point. On account of the possibility of hemorrhage the central end of the lateral sinus was lightly packed with iodoform gauze. Another reason for discontinuing the operation at this point was the fact that in two previous cases of extensive sinus thrombosis we had been forced to stop the operation before the thrombus had been entirely removed. Both of these cases recovered without further clinical symptoms, although in one of them the internal jugular vein was ligated at the clavicle, leaving a perfectly evident thrombus in the cardiac end of the vein.

The subsequent course in the case we are reporting proved that the patient had a cerebellar abscess in addition to the sinus thrombosis. This abscess was drained and he subsequently died with a diffuse suppurative meningitis. At the time of the operation we could find no evidence of an abscess definite enough to warrant an exploratory puncture. He had a choked disc and a slow pulse, and it was noted at the operation that the dura exposed over the tempero-sphenoidal and cerebellar lobes was quite tense, but it was

hoped that this was due to a serous meningitis. A lumbar puncture was not done at the time of the operation because there were no symptoms of a diffuse meningitis, and on account of the possibility of transforming a localized infection into a diffuse process.

Although the blood culture and those taken from the sinus at operation were reported as sterile, clinically the case was similar in every way to those in which a pneumococcus, type III, or a capsulated streptococcus has proved to be the infecting organism.

Immediately following the operation just described there was the same apparent improvement as after the two former operations. This, however, continued only three days, when the pain and stiffness in the neck was again complained of and about noon of the fourth day the patient complained of a chilly feeling; this was followed by a rise of temperature to 102° for the first time since the third operation.

The wound was free from pus but the tenseness of the exposed dura described by Dr. Crowe was very evident. The swelling of the optic nerve had increased slightly in both eyes, but more so in the left (the opposite side). The blood count showed a leukocytosis of 28,000 and the general condition seemed worse in every way.

This led me to suspect an extension of the thrombosis to the opposite side, but following the method of Dr. Crowe (which, by the way, is the most satisfactory demonstration of practical diagnosis I have ever seen) it was clearly demonstrated that there was no obstruction to the return of venous blood on that side.

Therefore, on the fifth day the packing was removed from the right lateral sinus liberating a perfectly sterile dark serum but no pus, a ring curette was introduced until the clot was reached and then gently rotated, with the removal of some colorless clot debris but nothing else.

On the following day the curette seemed to engage in a small opening in the sinus wall, from which a considerable amount of dark venous blood, followed by a small amount of pus, escaped.

The wound was dressed daily and about one cc. of pus could be expressed from the open sinus at each dressing. This drainage seemed to bring about a very encouraging improvement, the daily rise of temperature and the leukocytosis being greatly reduced and for nearly a week the patient complained of his neck only at night, being promptly relieved by one-tenth gr. morphin given about bedtime and would often sleep ten to twelve hours in the twenty-four. About this time, however, the patient began complaining of frontal headache and exhibited evidence of mild delirium at times, also

complained occasionally of chilly feeling, and for the first time in the course of the disease the stiffness in the neck could be demonstrated objectively and the patient was clearly losing ground. The swelling of the optic nerve on the right (affected) side had not increased, but the nerve on the opposite side was very much swollen although the lateral sinus and jugular vein on that side remained unaffected. On February 21, fifteen days after the last operation, the left eye exhibited all of the symptoms of cavernous sinus thrombosis, which continued to the end. On February 23, seventeen days after the last operation, I succeeded in evacuating under local anesthesia, a cerebellar abscess which had been discharging through the opening in the right lateral sinus wall for about ten days. This relieved instantly the tense pulsating dura described above but accomplished nothing more, the patient succumbing to a septic meningitis five days later.

Unfortunately an autopsy was not obtainable; it is clearly evident, however, that either the cavernous or the petrosal sinus (probably the cavernous) of the opposite side became involved toward the end; by what process of extension this was accomplished, I am entirely at a loss to explain.

It also seems clearly evident that the thromboses remained nonseptic to the end, the septic focus being limited to the cerebellar abscess, which must have been present from the beginning.

In this and similar cases the gravity of the disease is so entirely out of proportion to the clinical symptoms that a positive diagnosis is extremely difficult and often impossible.

The outstanding diagnostic symptoms in this case were the persistent though not constant complaints of discomfort in the back of the neck, the persistence of a high leukocytosis, the occasional chilly feeling and later the positive choked disc, the latter symptom appearing about the end of the fourth week.

The relation between the sinus thrombosis and the cerebellar abscess can only be conjectured. However, I am inclined to believe that the two processes were entirely distinct, though interdependent, as the thrombosis was a nonseptic process, while the abscess produced pus that was distinctly septic. The specific organisms being an unidentified Gram positive bacillus and a small streptococcus hemolyticus.

In closing I want to pay tribute to Dr. Crowe's method of demonstrating the presence or absence of obstruction to the outflow of blood from the brain. I know of few diagnostic procedures that give such definite findings.

BRAIN ABSCESS OF OTITIC ORIGIN WITH EPILEPTIFORM ATTACKS. DECOMPRESSION FOLLOWED BY RECOVERY.*

Dr. Carl Marion Sautter, New York City.

W. C., 13 years old, rated as a first-year high school student in a private school, became ill during the early part of January, 1920, with para-typhoid. While convalescing he was brought in contact with a case of measles. Shortly after this our patient also developed the measles, and had apparently recovered from this when he complained of a severe pain in the left ear. His physician within a few hours called in consultation an aurist. An acute suppurative otitis media of the left side was diagnosed and an incision of the drum performed. Irrigation with boric acid solution was made every three hours. On the fourth and fifth day the patient became very nervous, restless and suffered some from insomnia. His temperature range was from 100° to 102.5° F.

February 18 the writer was asked to see the case by Dr. Alexander Schmitt. His temperature at this time was 103° F. He was extremely nervous, apprehensive and rather septic in appearance. The right ear was perfectly normal. The left ear showed a profuse yellow discharge of pulsating character. It was difficult to keep the canal wiped. The incision made previously appeared to be quite extensive and afforded all the drainage possible. At this time slight tenderness was elicited directly over the mastoid antrum. A smear was taken and later reported to me as a pneumococcus. A specimen of urine was negative for albumen and sugar.

The nurse was instructed to irrigate the ear every hour with boric acid solution and even at intervals during the night. The case was seen daily and the canal cleansed. In spite of the utmost care the discharge remained, pulsating and very profuse, although there was no sagging of the drum. His insomnia persisted. On the 21st his temperature range was from 100° to 105° F. and repeated the following two days. The tenderness had increased over the mastoid, extending well over the temporal and zygomatic regions, but none over the tip. (Any increased tenderness over the meningeal area is significant of operative interference, especially with a ten-day history of aural discharge.) At this time a simple mastoid

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operation was advised. The same day an X-ray was taken at St. Luke's Hospital. This revealed an increased cloudiness of the left side and a most unusual extensive pneumatic mastoid bone. The cells were extremely high over the zygoma and temporal plate; the tenderness corresponding exactly to the location of these cells.

The patient was removed to the Misericordia Hospital, and a simple mastoid operation performed. The cells were quite numerous and extensive, especially in the middle fossa and overlying the temporal bone, where they were more superficial. Free pus was found in these with granulations but very little in the mastoid tip. The cells were thoroughly curetted to the plate, but neither sinus nor dura exposed.

The following day the temperature receded to normal and remained normal, and the patient was apparently making an uneventful recovery. The mastoid wound was looking fine with a diminution of discharge and no discharge from the canal. However, on March 4, that is, ten days from the date of the operation, the writer was informed that the patient was having a convulsion. Upon arrival at the hospital an hour later the boy was still in convulsions. He was in a deep coma with respiration only at great intervals and then a convulsion which was more or less generalized. The patient had been fine until about an hour previous to the attack, when he tried to ask for the urinal bottle, which he seemed to recognize but could not name. No history of chills or headache. The respiration and pulse rate normal. There was some difference of opinion as to whether the convulsions in the beginning were more right-sided than left-sided. There was no history of any former attacks of epilepsy and the family history was negative. Oxygen was given while getting the operating room in readiness. With the history of the word aphasia symptom and the convulsions probably due to a local meningeal edema, I performed a decompression. There were no adhesions of the dura, but a slight discoloration, although my light was poor at this time, due to the urgency of the case. The middle fossa was uncovered and the temporal plate removed about an inch in diameter, extending directly over the ear. A vertical linear incision was made through the dura about an inch in length. This released what appeared to me as coffee ground blood with a small piece of organized clot or sluff. A grooved director was passed forward, inward and backward in the brain substance in the various directions in the effort to locate an abscess, but with no success. A cigarette drain was inserted and the wound left open. In the afternoon of the same day the patient had apparently re-

gained his normal self. His reflexes were normal and equal. His conversation was coherent and rational. No aphasia or ocular nystagmus. At this time Doctors Dench and Garretson were called in consultation. Examination revealed: Fundi, negative. No nystagmus. All reflexes normal. Both upper and lower extremities slightly exaggerated but normal. Abdominal and cremasteric reflexes, normal and equal. Muscle tonus, normal throughout. Sensation (tactile, pain, temperature and muscle sense), normal. Examination of the right ear at this time proved to be normal. Urinalysis was made and was negative. Examination of spinal fluid and blood was made, showing normal cell count and negative Wassermann. At the end of four days patient continued normal with no temperature. The drain was completely removed at this time, having previously been shortened at successive dressings. There was no pus or exudate coming from the wound.

On March 10 in calling on our patient in the morning there was a distinct hesitation in his speech, rather stumbling in character with irrelative and monotonous phrases. He spoke of seeing his father, whom he had never seen, the latter having died shortly after his birth. There was no nystagmus, over-pointing or stiffness of neck. Reflexes were normal. No deviation of the tongue or twitching of the mouth. The condition remained about the same during the day except the continued motor aphasia with an inclination to vomit. However, at 6 o'clock the same evening they informed me that he was again having convulsions. About forty-five minutes later I reached his bedside. The patient was having convulsions at intervals but not in the deep comatose state that he had been at the previous attack. His contractions were quite evidently right-sided, involving the face and the limbs. He was immediately removed to the operating room, where I extended the removal of the temporal bone on a line with the supra orbital ridge and about one inch in front of the ear. A new vertical linear incision was made and a grooved director passed forward and downward about an inch towards the frontal convolutions. At the second passing a teaspoonful of rather blood-stained pus was evacuated. A cigarette drain was placed. The next day the temperature was about 101° to 102° F. Reflexes all normal and patient fully oriented. The second day the temperature was still at 102° F. In the morning when the drain was removed a considerable amount of thick exudate was found behind it. Instead of replacing the cigarette drain a small piece of rubber was rolled and inserted at the base of the abscess; following this the rubber drain was

removed twice daily and patient's temperature dropped to about 99° F. About this time he developed pain in the eye. Dr. C. Berens, Jr., was consulted and reported vision 20/15 and normal fundi. Two days later he developed another slight rise in temperature, which was due to the development of an abscess beneath the temporal muscle. This was readily drained, and his temperature dropped to normal and remained so until his complete resolution April 19. No hernia had developed in this case and his hearing was 20/20. Up to about a week ago when I last heard from this patient he was in good health and apparently quite normal.

Acute brain abscess cases we know very rarely complicate acute otitis media, and even less so the epileptiform attacks. If an abscess has been located and drainage established and maintained, resolution is the natural course, if located early enough. Of course there is always the possibility of the abscess rupturing, inundating the ventricles or the sub-arachnoid space, causing a suppurative meningitis. At the first decompression the symptoms pointed more to an abscess or pressure of the sensory speech center of the left temporo sphenoidal lobe, while the distinct motor speech symptoms prior to the second attack made it quite evident that further involvement existed more forward in the frontal lobe.

It seems to me that the simple rolled rubber drain proved most effective in maintaining the drainage. This of course was gradually shortened, allowing granulations to fill in from the bottom and to wall off the meninges.

11 East 48th Street.